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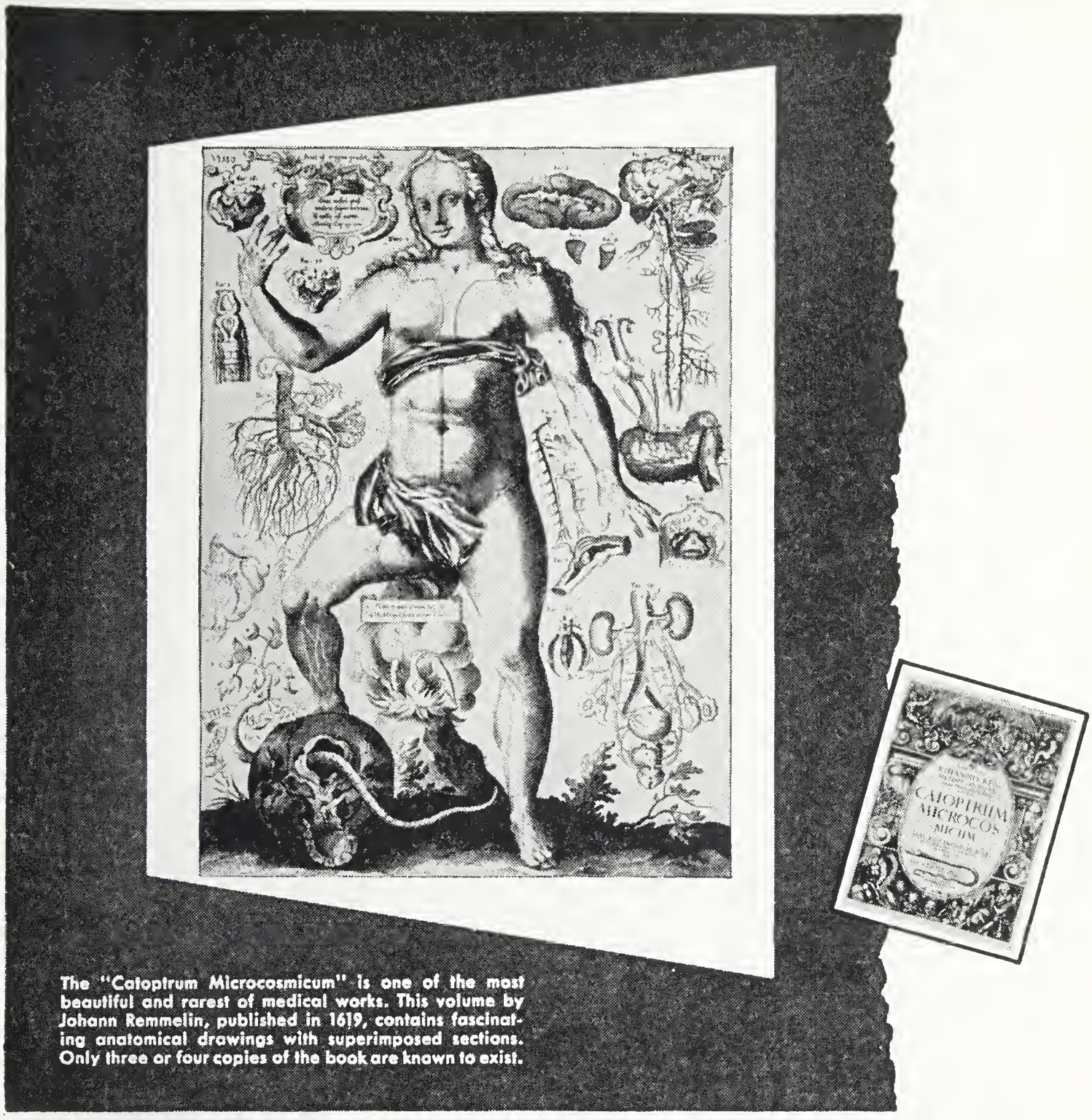

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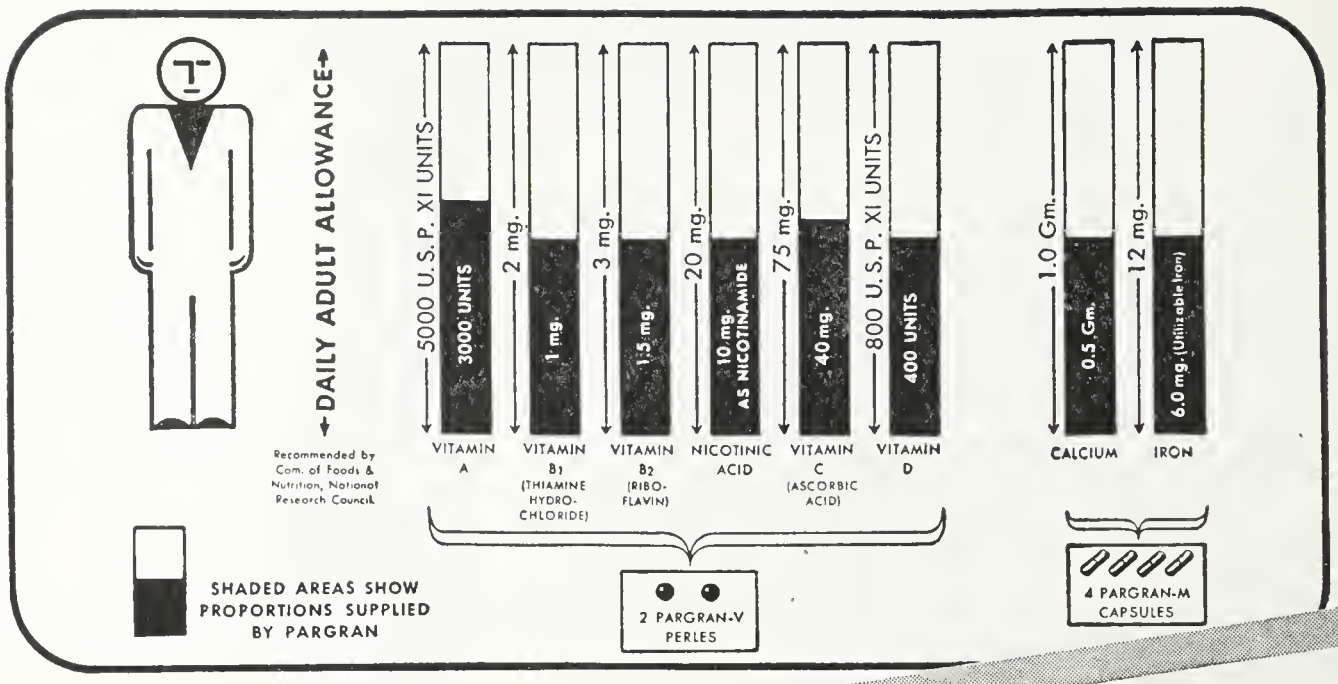

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Volume XX Toronto, September-October, 1942 Number 1

The University of Toronto Medical Journal

(AN UNDERGRADUATE PUBLICATION)

This journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

$1.50 per volume. 25c. per single copy.

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Editorial

At the present time, due to the exigencies of war, the fate of The Medical Journals hangs in a balance. In a university curriculum adjusted to wartime needs, neither graduates nor undergraduates are able to devote as much time to the preparation of material for publication as in the past. Nevertheless, the editorial staff has had considerably greater success in obtaining articles for publication from graduates than from undergraduates.

This should not be so. The nature and function of this Journal is essentially that of an undergraduate publication, and should be dependent, primarily, upon the student body and not upon the graduates, for its existence. Furthermore, certain graduates have expressed the opinion that what little time they have for writing, ought to be spent in writing their own articles for publication in general or specialized medical journals.

It should not be necessary for an editorial staff to be forced to seek articles. However, in view of the fact that very few articles are contributed, it has been necessary to go in search of material with which to make up a Journal. In addition, it has fallen upon the editorial staff to contribute in increasing measure to the pages of the Journal.

The editorial staff therefore feels that the Journal is a luxury which the student body enjoys at very small cost, and that if they wish this, *their* Journal, to continue publication, then, the undergraduates can best demonstrate this desire by contributing material for its pages.

THE INFLUENCE OF EXPERIMENTAL STUDY ON CLINICAL PRACTICE IN INTESTINAL OBSTRUCTION

ROSCOE R. GRAHAM, M.B.

The history of intestinal obstruction shows it to be one of the most lethal diseases. While progress has been made in the management of other diseases, it is only within the last decade that any lowering of the mortality in intestinal obstruction has been accomplished.

It is important to designate and recognize the various types of obstruction. These can be placed in three main groups:—

(1) Simple occlusion
(2) Strangulation obstruction
    (*a*) Closed loop
    (*b*) Mesenteric thrombosis
(3) Neoplasm
    (*a*) Rarely small bowel, unless secondary
    (*b*) Most cases large bowel

We of this generation of surgeons have no direct knowledge of the progress and fate of an undiagnosed and untreated intestinal obstruction. We recognize that the higher the site of the obstruction, the earlier death ensues. Few of us realize, however, that an individual may suffer an obstruction of the terminal ileum, and yet survive as long as two or three weeks without any treatment whatsoever. We do know, however, that in the cases admitted five or six days after the occurrence of a low small bowel obstruction, our best efforts of management pre-operative, operative and post-operative are followed by a staggering mortality within hours. This needs to be explained. We do know that the lower in the gastro-intestinal tract that a closed loop obstruction occurs, the more lethal it becomes, as opposed to the simple occlusive obstruction, in which the higher the site of the obstruction, the higher is the mortality. One must necessarily conclude that the seriousness of a strangulation obstruction is in direct ratio to the type and degree of bacterial content in the obstructed loop: the greater the infectivity of the content, the greater the mortality.

What are the known factors which are responsible for the clinical picture in intestinal obstruction? It is indeed more than the failure of passage of the intestinal content. We are all familiar with the patients suffering from habitual constipation, with very infrequent stools, and yet no disability whatever. The sequence of events which occurs in simple occlusive obstruction is distention of the bowel proximal to the site of the obstruction. As a result of this distention, there is a reversal of the normal physiological process in this segment of bowel. There is a decrease in the absorption and an increase in the secretion. Accompanying this there is increased peristalsis, resulting in vomiting, which is responsible for a marked fluid and electrolyte loss.

The degree of physiological and biochemical disturbance which follows a high obstruction is greatest and most easily appreciated when the site of the obstruction is in the pylorus. The sequence of events there is as follows:

(*a*) Distention of the stomach.
(*b*) Dehydration. In this case the fluid loss is due to vomiting.
(*c*) Demineralization. The chloride loss is very great, as well as a disturbance in the sodium and potassium.
(*d*) Alkalosis, which we recognize by the high CO^2 combining power of the blood.

(*e*) Azotaemia, in which the nitrogen retention is estimated in terms of the N.P.N. of the blood serum.

We have been conscious of this sequence of events in all cases of intestinal obstruction, the degree of the disturbance varying with the site of the obstruction. We have, however, probably been lost in a morass of biochemical and physiological detail, to such an extent that we have failed to realize that in simple language the patient is really starving to death. He lacks food. We have been conscious of the water and carbohydrate loss for many years, and have corrected this by the parenteral administration of glucose, saline and salt, but have we been alert to the significance of the associated protein loss? I think not.

When Hayden and Orr in 1925 presented their work showing the significance of chloride loss, and the presence of alkalosis in the presence of obstruction, our hopes were raised that the administration of large quantities of water and salt and glucose would make a marked contribution to the decreased mortality in this disease. It did, however, achieve much in making the patients more comfortable, but as the years went on we were disappointed to find that we had made very little impression on the total mortality. We realized that in this treatment, while one was able to restore the salt balance and bring the CO combining power within normal limits, the non-protein nitrogen of the blood remained constantly elevated until the obstruction was relieved, at which time it promptly fell to normal. Many people have misinterpreted the high N.P.N. and the decreased urinary output as indicating structural renal disease, and have accepted this as an alibi when their patient was progressing badly, and thus were lulled into a period of hopelessness. This disturbance of the kidney function, with the resulting nitrogen retention is a common, constant picture of intestinal obstruction. It is biochemical in origin, with no basis in organic structural renal disease. It is unfortunately impossible clinically to determine in all cases whether the patient be suffering from simple mechanical occlusion or a strangulation obstruction, because even though the major lesion be a strangulation obstruction, there is the associated disturbances of a simple mechanical occlusion accompanying it. It is, however, of the greatest importance to recognize that there is a basic difference in the character of the fluid loss in the occlusive, as opposed to the strangulation obstruction. While it is true that the circulatory blood volume is decreased in both, this volume loss in simple occlusion is due to loss of blood plasma, without loss of cells, and thus has an accompanying haemo-concentration, evidenced by a high haemoglobin. In the strangulation occlusion, because of the vascular involvement, there is in addition to the plasma loss a cell loss up to fifty per cent of the total. This produces a physiological upset comparable to that suffered by a patient who has had a massive haemorrhage which reduces the haemoglobin to 50%. Thus in all cases of obstruction there is a marked protein loss. The theoretical ideal in this disturbance would be plasma in the occlusive obstruction and whole blood in the strangulation obstruction. However, the recent work of Dr. Charles Best and his associates in our Department of Physiology suggests that plasma or serum is quite as efficacious as whole blood in restoring circulatory volume loss, even though

that volume loss be due to a massive haemorrhage. It is the protein which is needed.

The recent work of Professor Cohen of Harvard, in which he actually takes blood apart into its simple components, has thrown some light on this finding. He isolated the albumin fraction from the serum protein and found that a small volume of serum albumin introduced into the circulation acts as a cement, and prevents the transudation of fluid from the vessels into the interstitial tissue spaces: thus the introduction of a small amount of albumin, plus an intravenous of saline, would restore and maintain an adequate circulating fluid volume which would tide the patients over the crisis. That this hypothesis is true was substantiated by Ravdin, who left on the afternoon of December 7th for Pearl Harbor with human serum albumin, and his experience in treating the wounded of this engagement corroborated the experimental data of Professor Cohen. The small volume of albumin needed, and the ready availability of normal saline, makes this a real contribution in cases of emergency such as produced by the war. It rests now for the perfection of a method to produce this albumin so that it will become more readily available. It is also interesting to have a confirmation from Brigadier Ogilvie, of the Middle East Command, on the use of serum and plasma in the wounded, in which he has suggested in a recent letter that he believes blood in post-war therapy will become an obsolete fluid.

The practical clinical point to accept from these studies is that whether we are dealing with a simple occlusion or a strangulation obstruction, there is lost to the individual a large volume of protein which must be restored either by blood or plasma. Recently we have been utilizing the restoration of protein lost by giving Amino acids in the form of Amigen, which is the result of hydrolized casine.

One further clinical point is to recognize the tremendously increased cell volume loss in cases of strangulation obstruction, with the resulting severe anaemia, which demands therapy to correct the handicaps of the anaemic state.

There are certain facts which still leave us in doubt as to their explanation. All have seen the strangulation obstruction adequately fortified pre-operatively, operated upon with a negligible trauma, do well for twelve hours, and then die, with a falling blood pressure, rising pulse rate, pallor and marked listlessness. This brings up the possibility of a toxic factor in intestinal obstruction. The experiment of Foster and Hausler throws some light on this aspect. In an experimental animal they isolated a segment of bowel, restored continuity of the gastro-intestinal tract, and surrounded the isolated bowel with a rubber bag. Into this rubber bag collected the transudate from the closed isolated loop. The source of this transudate of course was from the circulating fluids, and was preceded by a marked oedema in the bowel wall, and in clinical practice we know that there is a marked transudate free in the peritoneal cavity. If the transudate collected in the rubber bag surrounding the isolated segment of bowel be injected into an otherwise normal animal, it produces toxic symptoms and a clinical state similar to those found in intestinal obstruction. The toxaemia which is in this transudate must result from tissue metabolism, plus bacterial action, and in the experimental work of these investigators, they

found that the more infective the material in the closed loop, the more toxic was the transudate. The relationship of the bacterial content of the loop to toxaemia is beautifully shown by transplanting a closed loop of gut in a new-born guinea pig into the peritoneal cavity of another animal. Nothing happens—no toxic factor about transplanted tissue autalyses and disappears. If, however, a similar closed loop from an adult guinea pig be transplanted, the host animal dies promptly, and at autopsy the cause of death is not peritonitis. These experimental facts explain two clinical pictures—first, the patient with the strangulated hernia has not the constitutional disturbances of a similar strangulation obstruction within the peritoneal cavity, this because the sac of the inguinal hernia is incapable of the degree of absorption which takes place in the peritoneal cavity: secondly, it may be the explanation of the marked improvement which is seen in a patient with a large bowel obstruction following the doing of a blind caecostomy which is accompanied by the outpouring of a large volume of intra-peritoneal exudate. The obstruction has not in the least been helped, because the caecostomy is not opened, on our Service, for at least twelve hours, but the clinical improvement in the patient is astonishing, and it is just possible that it may not all be due to the intravenous administration of fluid, salt, glucose and protein.

It has been suggested that this toxic substance may be histamine or choline, but assays have failed to reveal their presence in sufficient quantities to be significant. If this transudate contains a toxic factor, we must not lose sight of the fact that the fluid in the bowel wall producing the oedema of the bowel must also contain a toxic factor. However, during the study of strangulation obstruction, the interference with the circulation is so great that absorption does not occur. However, when the distention is relieved, rapid absorption takes place. This is beautifully proven in an experimentation by Braun and Borattau. They introduced a lethal dose of strychnine into an isolated closed loop of bowel. The animal suffered no ill effects whatsoever. If, however, the loop were suddenly decompressed, the patient dies of strychnine poisoning as quickly as if the drug were administered intravenously.

Wangensteen has proven that this toxic material does not reach the systemic circulation via the lymphatics, as he isolated and drained the thoracic duct, and material collected was completely innocuous. This data suggests that the aspiration of distended loops of bowel through an operation, together with the manipulation necessary to empty them, is bad, because of the possibility of releasing this toxic exudate into the blood stream. Manipulation of the bowel as is sometimes carried out by introducing a sucker into the lumen of the bowel and threading on the proximal distended loops, as originally suggested by Moynihen, would be the ideal procedure to release an abundance of this toxic material into the circulation, and thus is to be absolutely condemned.

One must then debate the wisdom of returning into the peritoneal cavity the loop of bowel which was the seat of the strangulation obstruction. Experience has almost universally shown that when there is any doubt as to the viability of a gut, it can be returned to the peritoneal cavity with safety, and while in this group of cases the mortality is high, one will at autopsy very rarely find any

perforation of the involved segment of bowel, nor will there be any evidence of peritonitis. Could this be explained by the absorption of the toxic exudate following the release of the obstruction? If this is a possibility, then we ought to debate whether resection of the obstructed loop would not be ideal: indeed this is sound practice, and was advocated by the late Sir David Wilkie. Whether a resection of this loop be undertaken will be determined by the environment in which the surgeon has to work, together with his experience, and that of his associates. The resection and immediate anastomosis of such a segment of bowel can be made safer if the anastomosis be carried out by incorporating a rubber tube across the suture line, as advocated by the late Dr. F. N. G. Starr.

This represents our clinical application of experimental data up to about five years ago: yet in 1933, in our own Department of Physiology, Professor Norman Taylor did a magnificent bit of experimental work, in which he showed the disastrous effects of distention, which is one of the early accompaniments of intestinal obstruction: yet he failed to make an impression upon the clinicians.

In the first experiment he proved, as has been done previously, that the distended isolated segment of bowel in which restoration of the continuity of the gut was accomplished resulted in a clinical state with signs and symptoms and death duplicating a strangulation obstruction. He then placed a rubber tube surrounded by a balloon in the gut. This did not in any way cause obstruction to the flow of intestinal content. Distention of the balloon, however, in the absence of obstruction to the passage of the content, produced a clinical state and death, as in mechanical obstruction. He then isolated a segment of proximal jejunum, and completely denervated it, and used it as a closed loop. This denervated closed loop did not give as sudden a death as the non-denervated loop. In other words, he concludes that the increased violent peristalsis associated with intestinal obstruction is harmful. This would suggest that we use large doses of Atropine. It was suggested that the bacterial content of the loop was the lethal factor, so an isolated segment was prepared, one end of which was left open until cultures revealed it to be sterile. The end was then closed, and the patient promptly died. These experiments were at the same time confirmed by Herrin and Meek, working quite independently of Taylor and his associates, and they both pointed so definitely to the disastrous effect of distention. However, no progress was made until the experimental physiologists again wanting to study the disturbance of metabolism in proximal jejunal loops, developed a double lumen tube, with which you are all familiar, under the name of the Miller-Abbott tube. These two investigators soon realized that if distention were of much importance in contributing to the mortality of intestinal obstruction, that this would completely solve their problem. This was supported by the use of the ordinary duodenal suction, as advocated by Wangensteen, but while the Wangensteen continuous suction will prevent distention in a large percentage of cases, it requires the double lumen tube passed beyond the pylorus to relieve it, once it has occurred. If this factor of distention be as important as Taylor suggests, then the use of the Miller-Abbott tube in addition to our previous thera-

peutic management should revolutionize the mortality in intestinal obstruction by greatly decreasing it. This would appear to be true. A survey of collected cases in literature confirms this:—

Mechanical Obstruction

306 cases—no suction—53 Died—17.3%
151 " —with " —12 " — 8.0%

Obstruction with Peritonitis

56 cases—no suction—41 Died—73.1%
52 " —with " —13 " —25%

Brigham Hospital Series
Mechanical Obstruction

56 cases—no suction—8 Died—14.3%
23 " —Colon obstruction—no suction—7 Died—30.4%

Obstruction with Peritonitis

36 cases—no suction—23 Died—63.9%
7 " —with " — 2 " —28.5%

Ward "A" Series—1930—38 inclusive
Mechanical Obstruction

30 cases—no suction—17 Died—56.6%
1939 to date
14 " —with " —14 " —28.5%

It thus becomes obvious that the improvement in the mortality rate confirms the fact that distention is a very important factor in the mortality of intestinal obstruction. However, such a revolutionary aid to treatment cannot be without pitfalls, and such is the case. We must realize that tube and suction treatment is of no value in cases of strangulation obstruction. The patient's condition may be improved because we have corrected the mechanical occlusive factor, which is an inevitable accompaniment of all strangulation obstructions, but this improvement should not lull us into inaction. This improvement in the mechanical occlusive obstruction treated by tube and suction is accomplished because relief of the distention abolishes pain: hence the patient can rest, the circulation in the distended segment of bowel is improved, absorption replaces secretion, relief of distention permits free movement of the diaphragm and allows also filling of the splanchnic vessels. Thus we must stress again the fact that, while the patient's condition may be improved, and he appears very comfortable, we must not be lulled into a state of false security, because even with such, he may be suffering from a lethal obstruction. In other words, the tube and suction treatment of intestinal obstruction completely eliminated the signs and symptoms which we have been taught to be an inevitable accompaniment of such serious disease.

There are two factors, however, which will help us to avoid this pitfall:— (1) Despite the improvement in the patient, we will not have decreased the non-protein nitrogen in the blood to normal limits. (2) The volume aspirated from the indwelling tube is not decreased, and is in large volume, and brownish and foul-smelling, and always in excess of the real intake until the obstruction is relieved. Thus we must remember that in the mechanical or strangulated obstruction, the tube is simply another means of improving the patient's pre-operative state, and there will be very few cases in which it will eliminate the necessity for operation, unless the obstructing factor be a peritonitis.

The method which we consider advisable in using the Miller-Abbott tube may be detailed as follows:—
(1) Anticipate distention in the infected cases, and intubate before the distention occurs. This will be accompanied by a passage of the tube through the pylorus in less time than if it is not utilized until distention has occurred.
(2) We are convinced that one indi-

vidual in each hospital should be assigned to the supervision and introduction of Miller-Abbott tubes.

(3) The capacity of the balloon on the end of the tube should be measured, and its volume recorded, before the tube is inserted, and at no time during the utilization of that tube should the volume be increased, because it is heart-breaking to have the tube just through the pylorus, and then rupture the balloon by over-distention.

(4) We have found it more satisfactory to detach the balloon, pass the tube through the nose, and then re-attach the balloon, before inserting it into the stomach. As soon as the balloon reaches the stomach, the contents should be aspirated, so that we are dealing with an empty stomach. Then we should be careful not to introduce sufficient length of tube to permit it to coil up within the stomach and we should not strap the tube to the patient's nose or forehead.

(5) The patient then lies on the right side. Continuous suction is maintained, and the tube is passed at the rate of six inches every half hour. We should avoid too frequent X-Ray observations, as in one case this resulted in a skin burn. One can tell when the tube is out of the stomach, because as long as it remains in the stomach, water taken by mouth will be immediately returned through the tube, which of course is not true if it has passed the pylorus.

One then adds a new problem when continuous suction is maintained in a tube lying in the upper jejunum, in that there is a tremendous loss of fluids and electrolytes. Therefore we must restore and maintain an adequate fluid balance. Most cases of obstruction on admission are suffering from chloride deficiency, and we give 500 c.c. of 5% sodium chloride intravenously, followed by 5% glucose in distilled water. There is a daily basic requirement of ten to fifteen grams of salt, depending upon the size of the individual. This is administered by the intravenous route of 1,000 to 1,500 c.c. of normal saline. In addition there must be sufficient fluid to provide 1,000 c.c. of urine. This is given intravenously, using 5% glucose in distilled water. If, after hydration is adequately overcome, the haemoglobin is 70% or less and the red blood count 3,000,000 or less, then the patient definitely requires protein in the form of plasma, serum, blood or Amigen. One should also record the daily intake per os and per intravenous, and balance it against the daily output from vomiting, tube suction, urine, and discharge from fistulae. One must replace the total fluid loss, c.c. for c.c., with normal saline, and then add sufficient 5% glucose in distilled water to produce 1,000 c.c. of urine in 24 hours. The difference between the intake and the output will be the best guide as to the progress of the patient, because until the intake per os is much greater than the output per tube suction, we have not solved the patient's problem.

A further use of the Miller-Abbott tube should be mentioned. Often the operative procedure has to be terminated with resulting small bowel fistulae, from which the fluid and electrolyte loss is enormous. The tube is passed until it reaches the site of this fistula. Several things are then accomplished: (1) The fluid loss is accurately measured. (2) Irritating discharges are prevented from reaching the skin and excoriating it. (3) An adequate, well-balanced diet may be taken per os, with the resulting nutritional balance. (4) The operation which will be necessary to close the

fistula ismuch safer, and tube suction prevents any strain on the suture line.

We have stated that large bowel obstruction with abdominal distention is a contra-indication to the use of tube suction: yet the principle which guides the treatment of such patient is the same. Distention must be overcome. This is accomplished by a simple blind caecostomy. The same lethal toxic factor in the transudate and oedema of bowel wall is present in obstruction of the colon, but in view of the fact that it is in a segment of bowel laden with highly-pathogenic bacteria, it is much more toxic than a similar transudate in the small bowel, and any manipulation of the obstructed large bowel is much more disastrous even than the equally objectionable manipulation of an obstructed small bowel. This is the basic principle which guides us to do a blind caecostomy without an exploration. In large bowel obstruction, which is in the vast majority of cases due to neoplasm, we can add another aid, that is defunctioning by means of a Mickulicz type of colostomy in the transverse colon, as over 80% of the obstructing lesions of the colon lie distal to this site. This makes possible the safe and radical dissection of low colon carcinomas, and will avoid a permanent colostomy in more cases than we have in the past, and still give a very excellent survival period comparable to the series in which a permanent colostomy was done.

In conclusion

The basic and early disturbance of distention of the obstructed bowel holds the key to all the subsequent disturbances, physiological, biochemical and physical, which accompany intestinal obstruction.

The relief of the distention by means of a double lumen tube has offered the greatest advance by any single therapeutic procedure.

Combining this with the administration of fluid, salt, glucose and protein has decreased the morbidity and the mortality of this disease in a most delightful manner.

Protein may be administered as serum, plasma, or in the form of Amino Acids, by the administration of Amigen.

The lag which has inevitably occurred in the clinical application of experimental data should stimulate us to be constantly on the alert to decrease this lag to as great a degree as possible.

WHEN IS A HERNIA?

H. F. ROBERTSON, F.R.S.C. (C.)

In time of war many problems assume new importance. The diagnosis and differential diagnosis of inguinal and femoral hernia is one of these. In peace-time a patient with suspected hernia may be watched; the patient and the doctor can think over the advisability of operation. Usually there is no great urgency. But in war, the patient and all concerned become impatient with equivocation. Is there a hernia or is there not a hernia?

Frank inguinal hernia is frequently seen in young men of military age. But many herniae are small and incomplete and real difficulty may arise in deciding whether or not a small or incomplete hernia is actually present or not.

It is necessary to make a definite statement about the presence or absence of a hernia. On the one hand one does not want to discourage a young man preparing to join the Armed Forces, or to expose an individual to operation and many inconveniences by stating that a hernia is present unless the diagnosis is certain. But on the other hand, missing a hernia may mean eventually more inconvenience to the patient and everyone concerned. For later the hernia is sure to be picked up and many arrangements will be upset. Statements that intimate the possibility but not the probability of hernia, that hint at a "weak wall" or a "relaxed ring" are not acceptable. Such hedging leaves everyone disatisfied. If a definite sac is not palpable, in the inguinal or femoral canal, one should say so; any laxness of the rings or bulging of the surrounding structures should be recorded as additional information.

It is most important to follow a definite routine in the examination for hernia. Visual inspection often reveals a hernia which palpation misses. The patient should always be placed facing a strong light, the examiner facing the patient. Any bulging in the inguinal region is brought out clearly by having the patient turn his head to either side and cough. Coughing usually bulges the affected groin and external ring and if a hernia is complete it will usually descend into the scrotal region while under observation. Indirect hernia bulges obliquely and if complete goes into the scrotum. Direct hernia bulges forward and then along the canal, or bulges out of the external ring, but it usually does not go into the scrotum. Femoral hernia bulges below Poupart's ligament and lateral to the pubic spine.

When from history or for other reason hernia is suspected but not seen when the patient coughs, or if one is not sure of the findings when the patient coughs, have the patient hold the nose between the thumb and finger, close the mouth and bulge the abdominal wall strongly. This frequently will give a better picture of the true direction and extent of any local bulge of the abdominal wall. If now no hernia is definitely diagnosed and the presence of one is still suspected, have the patient jump up and down several times, then squat and cough; or, holding the nose between the thumb and finger and closing the mouth, distend the abdomen. This manouvre will expose a sac very often. Be sure by close observation in all cases that the patient is not hiding a hernia by tensing the abdominal muscles. This occasionally serves to mask a small hernia.

Lastly, under inspection, a word about a rather variable but frequently widespread and diffuse bulging in the inguinal region seen on coughing or straining. Unless palpation confirms a definite sac, hernia should not be diagnosed in these. A Frenchman, Malgaigne, about the middle of the 19th century, described this type of bulging lower abdominal wall, rather fully. He said there was no true hernial sac present but felt that most of these cases would later develop hernial sacs through the weakened abdominal wall. A good picture of this variation of normal anatomy is in Vol. 11 of the text of the London surgeons, Bailey and Love, 1932 edition. Such cases have been operated on for hernia and no sac found. Usually the bulge is due to a thinned out aponeurosis of the external oblique muscle and a conjoined tendon that does not hug the cord closely but tends to leave a large gaping internal ring. The transversalis fascia is often quite strong and serves to prevent true herniation. Most people nowadays agree that true hernia does not occur in these cases more often than in the perfectly normal young male adult.

Palpation serves to confirm and extend observations already made. Several points need emphasis. First, palpation of the inguinal region with the fingers over the external ring or over the femoral canal should always be done to confirm the presence and direction of any bulging seen. Then the little finger gently invaginates the loose skin about the upper scrotum into each inguinal canal in turn; care being taken not to cause pain by pinching the skin with the finger nail. The external ring varies considerably in size but a relaxed and poorly defined ring or one much larger than the midfinger should be noted. The normal canal on coughing or straining closes slightly on the fingers, the external ring tending to grasp the finger. In many cases the pulp of the finger notes constricting pressure or bulging of the posterior wall of the canal. This should not be mistaken for a hernial sac. A sac must be a definite isolated protrusion which comes either down the canal against the finger tip or into the canal against the finger from the posterior wall. A good plan is to see if the protrusion felt will push the finger along or out of the canal. Then see if the mass can be pushed back. To do this it may be necessary to have the patient lie down and relax the abdomen by flexing the thighs. When the suspected hernia has been reduced the fingers are held over the canal opening or a finger is inserted in the canal and the patient strains or coughs while one feels the for reappearance and the direction of the hernial sac. Large incomplete or complete herniae rarely are confusing and need this amount of examination. The sac is quite definitely seen and felt in front of the cord and only needs to be differentiated as to the type and whether or not it is complicated by the presence of a varix, a hydrocele or undescended testicle; and of course whether or not it is one of these that is actually present and not a hernia.

An indirect hernia is liable to have stretched the external ring to a size larger than that of the diameter of the little finger. However, sometimes a hernia is present with a small external ring. The canal is usually definitely relaxed. When the patient coughs an impulse is felt into and down the canal against the examining finger which is lying anterior to the cord. If the inferior epigastric artery is palpable, and it is rarely palpable, the hernia will

be found coming into the canal lateral to the pulsating artery. Direct hernia comes through the posterior canal wall medial to the mid-Poupart point and medial to the inferior epigastric vessel if it should be palpable. The sac protrudes directly forward and usually it does not come past the external ring. This is the so called bubonocele. Femoral hernia may lie over Pouparts ligament resembling an inguinal hernia but the inguinal canal is empty on palpation and if the hernia is reducible it can be traced to the femoral canal.

Having determined definitely the presence or absence of an abnormal mass in the groin, further observations including auscultation and percussion serve for differential diagnosis. Is it a hernia of bowel or omentum? Is there an undescended testicle present, a hydrocele or a varicocele?

Herniation of bowel presents a mass which is tender on palpation, gurgles and has an expansile impulse on coughing. The mass does not fluctuate. It is often resonant. It does not transilluminate except very occasionally. The last part of the tumor reduces easier than the first part; and after reduction the mass usually will not come down into the scrotum again if the fingers are held over the external ring while the patient stands. Coughing or severe straining bring the mass down against the finger where the above characteristics can be made out.

Herniation of omentum presents a doughly, slightly nodular, irregular mass which is usually not tender on palpation. There is no gurgling. There is little or no impulse when the patient coughs. The mass is dull on percussion and does not transilluminate. The first part of an omental mass reduces easier than the last part; but it is rarely completely reducible.

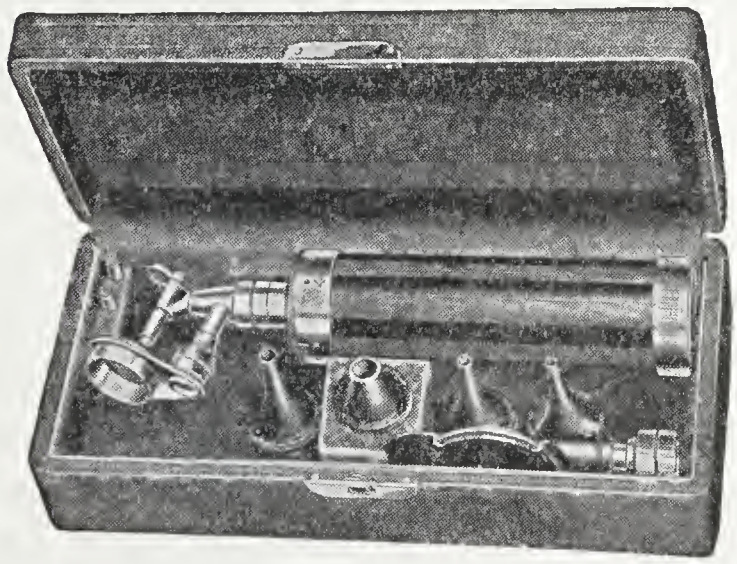
A hydrocele presents quite a definite set of findings. It is tense and elastic. It will not reduce unless it is of the congenital type. One can get above it with the fingers as a rule. If the testicle is pulled down the mass moves with the cord. The hydrocele is nearly always translucent. It is dull on percussion and is so tense that it does not fluctuate. If a hernia is present with the congenital type of hydrocele, on transillumination and palpation one can differentiate bowel, testicle and fluid mass.

A varicocele is a very common finding in young men, nearly always on the left side. The typical "bag of worms" is diagnostic as a rule. The mass is not painful on palpation. A fluid thrill is usually present when the patient coughs and also a bruit. Fluctuation can usually be obtained between one finger high on the mass and one finger low down. The mass is not translucent. The veins will disappear when the patient lies down and the scrotum is held up while the veins are compressed, but will reappear again when the patient stands even if the fingers are held over the external ring. Occasionally a hernia and a varix are present together. In such a case differentiation is made by placing the pulp of the finger on the cord in the canal. Then when the patient coughs one can feel the hernia descending against the one side of the finger while the thrill of the varix is felt by the other side of the finger. When a varix is very large in association with a hernia, the varix is emptied by the patient lying and elevating the scrotum. Then when the patient stands, the examiner's fingers being held over the external ring, the varix will fill first; when the patient coughs or strains the

hernial sac may be felt descending as a separate mass.

Undescended testicle usually does not present any problem because of the empty scrotum on the affected side. If the testicle is in the canal pressure over the mass will usually elicit testicular sensation unless marked atrophy is present. The mass of course will not be resonant and will not transilluminate. As hernia often accompanies undescended testicle it is important to make sure whether hernia is present. If the testicle is readily palpable it should be isolated by the tip of the finger and when the patient coughs or strains, watch for the appearance of a sac on the other side of the finger or at the finger tip, that is, in front of the testicle and cord.

In summary it is seen that the accurate diagnosis of hernia, as of anything else, depends entirely on a careful and thorough examination. Inguinal and scrotal masses will occasionally present difficulty in differentiation but accurate observations on the above points will do much to make the diagnosis self evident.



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TUMOURS OF BONE

G. A. LANE, M.D.

Introduction

A tumour is a growth of new cells which proliferates without control and which serves no useful function. Tumours of bone strictly speaking then, should be confined to growths of bone cells proper, but for practical purposes we must include tumours of certain other cells which, because of anatomical proximity, are linked with bone tumours clinically.

A proper understanding of tumours of bone is based on an accurate conception of all the cells present in normal adult bone, and more particularly on a knowledge of how those cells got there. In other words the embryological study of normal bone is more important for this purpose than a histological study. The next important principle to grasp is that the development of bone is not a process that occurs in utero and is an established fact by the time the fœtus makes its first public appearance, but is a process that continues actively right up to and beyond adult life, and is only complete when the last bit of pre-osseous cartilage in the body has been ossified. Thirdly, there is the fact that even when fully developed, bone far from being in a state of rest, is continually in a state of flux, being absorbed here and laid down there as occasion demands. True, such a process is very slow according to our standards, but it is none the less factual and must be remembered. Lastly we must take account of the possibility that embryonic tissue persisting in abnormal situations may give rise to tumours consisting of cells not usually found in adult bone. *It is in delayed developmental steps in the persisting primitive connective tissue of the skeleton and in conjunction with subsequent histiogenic steps after the cartilage of the skeleton has been formed that practically all primary bone tumours take origin.* Since it is the development of bone that is so important for our purposes it may be worth while to take the time to refresh our knowledge of this subject.

Development of Bone

Adult bone is classified as one of two varieties according to the mechanism by which it has come into existence. The two varieties of adult bone are (1) Endochondral bone, and (2) Membrane bone.

Endochondral bone is bone that has been first laid down in cartilage. The area in the fetus in which bone is going to form consists of embryonic connective tissue or mesenchyme. The undifferentiated mesenchymal cells first develop into chondroblasts and these cells lay down a mould of cartilage in the shape that the adult bone will assume in the future. This cartilaginous mould is the so-called blastima of the future skeleton. Outside this blastima, fibroblasts occur and form a membrane of fibrous tissue which together with the underlying mesenchymal cells, is known as the perichondrium. Interstitial growth occurs as this perichondrium lays down layers of cartilage on the original blastima. With the appearance of a richer blood supply in the region, the primitive mesenchymal cells differentiate into osteoblasts which now lay down a shell of bone around the cartilage model. The perichondrium has become the periosteum. The central cartilage next commences to break down and soon a

tuft of blood vessels finds its way into the centre of the shaft through the shell of bone. Osteoblasts accompany this periosteal bud, as it is called, and begin to lay down bone centrally. With the resorption of more cartilage, more bone is laid down until almost all the cartilage has been transformed into osseous tissue. A similar process occurring at each end of the bone forms two epiphyseal centres of ossification as opposed to the diaphyseal centre previously discussed. Until these centres unite they are separated by a cartilaginous disc, the epiphyseal plate, by means of which it is possible for the bone to grow in length. When these centres meet, the epiphyseal cartilage is ossified and growth in length is no longer possible. However, as previously mentioned, even when this occurs, appositional growth is still possible, and does occur; nor are all the primitive cells lost, as is shown by the readiness with which they respond to an adequate stimulus, e.g. a fracture.

Membrane bone differs in its formation in that it does not go through this cartilaginous stage, but is laid down as osteoid tissue which rapidly undergoes ossification to form adult membrane bone. It is not a more primitive mode of bone formation but a more advanced one in which the intervening cartilage stages are omitted. If all our bones developed similarly we would lose the source of many of our bone tumours because it is in these steps that they have their origin. Consequently we find that membrane bone is not commonly the primary site of a bone tumour.

Classification of Bone Tumours

A—Benign tumours related to bone formation: (1) Osteochondroma. (2) Multiple exostoses. (3) Chondroma.

B—Benign tumours related to bone resorption. (1) Solitary bone cyst. (2) Giant cell tumour.

C—Malignant tumours of osseous origin: (1) Primary chondrosarcoma. (2) Secondary chondrosarcoma. (3) Sclerosing sarcoma. (4) Chondroblastic sarcoma. (5) Osteolytic sarcoma.

D—Malignant tumours of non-osseous origin: (1) Ewing's tumour. (2) Multiple myeloma. (3) Fibrosarcoma and neurogenic sarcoma. (4) Secondary carcinoma.

Osteochondroma

Histogenesis—These tumours are considered to be an exaggeration of a normal bony protuberance intended for the anchoring of an important tendon. At such a junction nature provides normally for a protuberance of bone bulging through a gap in the periosteum to meet an adjoining tendon, which co-operates in the formation of the attachment by cartilaginous ossification within the substance of the tendon. These centres of proliferating cartilage cells in the tendon ends form part of the extra-skeletal blastima. The origin of an osteochondroma depends on a failure in the surrounding periosteal ring to close the gap between it and the cartilaginous centre in the tendon. As a result the cartilage is not replaced and keeps proliferating to form a protuberance of normal bone with an epiphyseal cap. Such a failure may be due to trauma, chronic infection, or may be a hereditary defect.

Clinical Features—From a consideration of their mode of development it is easy to see that osteochondromata must occur in the early decades of life (10-25), and that they must occur in the regions where important tendons are attached, i.e. near the joints. As the process is

merely one of an unbalance in a normal process not a reversion to a primitive function, such a tumour should not be malignant. Consequently these tumours are often undiscovered, and when they are found the only disturbance they cause is due to the size or the situation of the growth. The usual story is of the discovery of a painless swelling of the bone.

X-ray—This shows an osseous outgrowth with cancellous and compact portions, merging with normal bone beneath and capped with cartilage in varying degrees of calcification.

Pathology—The gross and microscopic appearance will obviously be indistinguishable from that of normal bone, because the tumour merely consists of normal bone in an abnormal situation.

Prognosis and Treatment—The tumour is quite benign and only requires excision when it presses on other structures or an overlying bursa develops. Particularly after 30 however it may undergo sarcomatous degeneration, indicated by a sudden exacerbation of symptoms or a sudden increase in size. In this case it must be treated as a sarcoma.

Multiple Exostoses

Histogenesis—This disease, also known as hereditary deforming chondrodysplasia, is due to a congenital disturbance of the perichondrium. This tissue fails to develop fully into periosteum and the effects are multiple. Because of the deficient periosteum the cortex fails to grow as thick as normally; the periosteum fails to limit appositional growth of bone effectively resulting in a widening of the metaphysis; and there is a failure to unite with the extra-skeletal blastima resulting in the formation of multiple exostoses as described in the preceding section.

Clinical Features — The underlying bone deformities cause visible deformities in the build of the individual, such as shortening and bending. These deformities are hereditary. There may be local signs due to the presence of an exostosis. The deformities tend to become stationary in adults.

X-ray — There are three features: (1) Typical exostoses. (2) Widened metaphysis. (3) Deformities (usually bending and fusion of tibia or fibula).

Pathology — The gross appearance corresponds to the X-ray picture and the microscopic is identical with that of the solitary osteochondroma.

Prognosis and Treatment—The prognosis for life is good, but there is no adequate treatment for the deformities except surgery for correction after the growth period is over. Malignant change is occasionally reported.

Chondroma

Histogenesis—For an explanation of these tumours we must return to the primitive cartilaginous blastima laid down in the fœtus. This blastima was stated to assume the shape of the future osseous skeleton. At first it is a continuous mass, but soon strands of precartilaginous connective tissue cut across the axis of the future bone and by a process of mucoid degeneration form the cavities of the future joints, while the lining cells form the future joint cartilage and synovial membranes. Occasionally however aberrant strands of this precartilaginous tissue occur and do not regress but remain to form islands of cartilage in the centre of the bone. As a result of future benign multiplication of these cells a cartilage tumour or chondroma is formed in the centre of the bone.

Clinical Features—From the mode of

origin we should expect the age incidence to be the same as in the other members of this group, which it is. It will also tend to occur near joints, but there will be a greater tendency for it to occur where joints are most numerous, as this is where there are most likely to be aberrant strands of joint tissue. Consequently this tumour is most frequent in the small bones of the hands and feet, and also at the anterior ends of the ribs. Most tumours are discovered in the decade between 20 and 30 and are manifested by a swelling in the hand or foot which slowly increases in size. Symptoms are mild and protracted. The growth is usually single and rarely involves the long bones, but when it does it has a greater tendency to undergo malignant change.

X-ray—The tumour appears as a small translucent rarefied area occurring centrally in the shaft of a small bone. The overlying cortex becomes thinned and expanded so that the picture is that of a large cystic cavity, surrounded by a shell of bone.

Pathology—The tumour consists of a mass of cartilage which appears as a lobulated and gelatinous growth. Numerous pockets are marked off by fibrous trabeculae, and contain translucent congealed material. Microscopically the tumour consists of cartilage in various stages of growth.

Prognosis and Treatment—Although these tumours are inherently benign, those occurring in the long bones and ribs particularly are quite prone to malignant change. The treatment depends on the location. Small tumours in the bones of the hand may be left alone unless local symptoms demand excision. Recurrence is unlikely following excision or curettage and cauterization. Tumours of the ribs or long bones on the other hand must be watched carefully for malignant change. Recurrence is likely following removal; complete excision with irradiation offers the best chance of cure.

Giant Cell Tumour

Histogenesis—Although this tumour is locally destructive and invasive, it is non-malignant. For an understanding of its origin we must return to the process of endochondral bone formation. This was shown to consist essentially in three stages: (i) calcification of cartilage to form primary areolae, in the shaft of the bone; (ii) invasion of these areolae and resorption of their walls, by vascular mesenchymal tissue; (iii) laying down of permanent bone, on the calcified framework left.

It is on stage (ii) that we wish to focus our attention. This is carried out by means of vascular mesenchymal tissue. From fusion of mesenchymal cells, giant cells or osteoclasts are formed. These may be for the purpose of causing absorption, or may be present merely as a result of absorption. For our purposes, distinction is unnecessary. Microscopic examination of this mesenchymal tissue shows it to consist of numerous blood vessels, osteoblasts, mesenchymal cells and giant cells—a picture identical with that of giant cell tumour. It is the contention of Geschicter and Copeland that giant cell tumour is nothing but this same process being carried out in adult bone, where for some reason the normal removal of calcified tissue has overstepped the bounds of normality and a tumour has occurred. They further postulate as the cause of this some interruption of the blood supply, e.g. by trauma. Interruption of the cortical blood supply, as by the formation of a periosteal

hematoma, suspends the activity of the underlying cortical bone. In compensation the medullary circulation is increased, but this new blood supply can only reach the avascular part after osteoclasts have opened up channels in the bone by a process of resorption. Now it must be remembered that the region of bone where resorption is most active in the young adult, is in the region of the epiphyseal plate. If then by the method outlined, osteoclastic activity in such a region is further increased while cortical function is temporarily suspended, the result will be a hyperplasia of osteoclastic tissue unchecked by the usual restraining influence of the cortex. In the metaphysis the cortical bone is thick and eventually resumes control. In the epiphysis however the cortical bone is much thinner and bone destruction occurs much more quickly. The result is that in the metaphysis we have a resisted lesion or a benign *bone cyst*, whereas by the same process in the epiphysis there is produced a *giant cell tumour*. Bone cyst is thus a healing reaction in normal bone that occurs through the activity of osteoblasts without the transitory stage of cartilaginous calcification. This reaction may be due to inflammation, foreign body, or tumour, and is a non-specific tissue reaction. Most cases occur around areas of bone destruction, and so perhaps many a bone cyst marks the grave of a beginning giant cell tumour. On the contrary too, a giant cell tumour constitutes a victory for the resorptive processes over the normal resistance of osteoblastic proliferation.

Clinical Features — This theory explains the age incidence, greatest from 15 to 25. The location at the extremities of the long bones is also a logical consequence of the mode of origin. The relation to trauma and bone cyst is obvious. The symptoms include the history of trauma, followed by swelling, pain, and pathological fracture.

X-ray — An asymmetrically placed area of bone destruction is found at the end of a long bone. The lesion begins subcortically and extends centrally. There may be a soap bubble appearance due to the presence of bony trabeculae. The overlying cortex forms a thin bony shell which is continuous with normal cortex. There is no periosteal reaction.

Pathology—The tumour consists of a hemorrhagic, friable, solid tissue and numerous small cysts with irregular bony or fibrous trabeculae. The bony capsule is very thin. Spread is rapid through cancellous bone, but the joint cartilage resists the growth of the tumour. Microscopically the tumour consists of giant cells with a loose cellular stroma containing round cells, spindle cells, and blood spaces.

Prognosis and Treatment — As was stated before the process is benign. Spontaneous cure may occur, or may follow a spontaneous fracture. A bone cyst may be the end result. Curettage is usually successful in those cases requiring treatment, although for inaccessible regions radiation may be preferable.

Primary Chondrosarcoma

Histogenesis—This tumour is related by structure and location to primitive precartilaginous connective tissue, e.g. a tthe site of tendon attachments. The occurrence of centres of proliferating cartilage cells in these situations has been referred to previously in considering the exostoses. Persistence of such tissue provides a normal growth centre that functions in maintaining tendon

length in keeping with increased skeletal growth. Such centres however may also undergo malignant change. This occurrence has been postulated as being due to tissue imbalance of some sort by which instead of a benign exostosis a malignant tumour is produced.

Clinical Features—As this is a case of change occurring in primitive tissues, this tumour is extremely malignant. It tends to occur at the period of maximal skeletal growth as this is the period at which the greatest strain is put on these growth centres. Most cases occur between the ages of 14 and 21. The situation is periosteal and periarticular. Corresponding with its degree of malignancy the course is very rapid, and the history is usually of about six months duration. Characteristically there is pain, which may follow trauma and is so severe as to interfere with function. This is followed by local swelling. Constitutional symptoms may occur.

X-ray — The characteristic features are: (1) A faintly visible, translucent, soft part shadow next to the bone. (2) Raising of the adjacent periosteum, with lipping. (3) Absence of cortical or medullary involvement so that the entire tumour is outside the bone.

Pathology—The tumour consists of a pearly gray shiny mass at the situations described. Microscopically it consists of cartilage undergoing cystic degeneration and is accompanied by a small amount of ossification and some cellular precartilaginous connective tissue.

Prognosis and Treatment—The tumour is unusually malignant and tends to invade the veins. It is not radiosensitive and is only cured by amputation in about 10% of cases.

Secondary Chondrosarcoma

Histogenesis — The histogenesis of these tumours offers no difficulty since by definition they arise from benign tumours. The persistence of connective tissue of the primitive precartilaginous type in the exostoses makes possible the occurrence of secondary chondrosarcoma superimposed on a benign growth. This tumour may complicate any of the benign growths or multiple skeletal disease such as Paget's disease.

Clinical Features—As this is a secondary degeneration it occurs in a later age group that of 35 to 55. Typically it is located in the ribs, upper humerus, or heel, as opposed to the primary type. Usually there is a history of a benign tumour present for years followed by a short history of acute symptoms.

X-ray — Radiologically the benign tumour can be seen and there is a superimposed malignant change, shown by an infiltrating periosteal shadow invading the benign lesion from above downwards.

Pathology — Cartilaginous masses interspersed with calcified and osseous material and large amounts of myxomatous connective tissue are found.

Prognosis and Treatment—The course is chronic. The lesion is radioresistant and can be cured by surgery in about 25% of cases.

Sclerosing Form of Osteogenic Sarcoma

Histogenesis—This tumour arises in the osteogenic layer of the periosteum. This layer is particularly active in the formation of compact bone in the metaphyseal regions of young adults. The sub-periosteal region of the epiphyses is not active after the age of three and hence is never involved primarily. Similarly with the midshaft region.

Clinical Features—This is the most highly differentiated of the periosteal sarcomas and hence can occur in membrane bone as well as endochondral bone. It occurs in the age group 15 to 25 when osteogenic activity in this region is at its height. The location is characteristic, being on the metaphyseal side of the epiphyseal line and in 80% of cases occurs around the knee. The duration of symptoms is brief. There is a sequence of trauma, pain, tumour, and dysfunction in many cases.

X-ray—Dense formation of new bone is found in the typical situation. There may be some secondary destruction in the marrow and there is new bone formation extending into both medullary and periosteal zones. The periosteum is raised and lipping occurs. Dense radiating spicules of bone give rise to the characteristic sun-ray appearance.

Pathology — As the growth is an exaggeration of the function of osteogenesis, the specimen consists of bone, often of extreme density, stripping the periosteum and invading the surrounding tissues. Microscopically there is proliferation of osteoblasts with irregular bony spicules and occasionally areas of cartilaginous tissue.

Prognosis and Treatment—The neoplasm is quite malignant and is radioresistant. Radical resection carried out promptly results in cure in about 25% of cases. If there are no evidences of tumour 18 months after operation the patient is likely cured.

Chondroblastic Sarcoma

Histogenesis—In the adolescent period of normal individuals there is at the epiphyseal lines of the long bones a final spurt of growth in length of the appendicular skeleton, just prior to the obliteration of the growth discs. On the metaphyseal side of the epiphyseal line there is chondroblastic proliferation and maturation of cartilage cells. This is followed later by vascularization and new bone formation. In malignancy this ordinary process is distorted and there is a definite imbalance in favour of the chondroblastic growth causing a tumour of rapidly proliferating chondroblasts.

Clinical Features—These tumours will occur between the ages of 15 and 20. They are localized to the regions of ossifying epiphyseal lines. There is a history of trauma, pain, tumour, and dysfunction, occasionally with general symptoms.

X-ray—There is an area of bone destruction in the typical situation. There may be a bony shell, of varying density and a periosteal reaction may occur.

Pathology—The picture resembles that of giant cell tumour from which it is not distinguished by some writers. The tumour invades bone and soft parts and contains remnants of hyaline cartilage, with chondroblasts and giant cells. The connection to the epiphyseal line can be traced.

Prognosis and Treatment—The tumour is rapid growing and fairly malignant. Occasional cases have been controlled by radiotherapy, but radical surgery and irradiation combined are usually indicated.

Osteolytic Sarcoma

Histogenesis—Osteogenic tissue occurs on both sides of the cortex, but the mode of ossification differs slightly. In endosteal ossification, calcified cartilage or else adult bone must first be removed before new bone can be laid down. The process is a more rapid and vascular one than the periosteal type. Malignant

disorganization of this process results in a tumour that tends to invade and destroy bone rather than form it as in the sclerosing osteogenic form of sarcoma.

Clinical Features—This tumour has a wide age distribution corresponding to its histogenesis and there is a corresponding variability of the region involved, although it is most frequent at the ends of the long bones. The characteristic history is one of pain, tumour, limp, trauma, and fracture. The duration of symptoms is about one year.

X-ray—There is a central area of irregular destruction which extends through the unexpanded cortex and causes a periosteal reaction. The cortex then expands slightly and is perforated.

Pathology—The tumour consists of a highly vascular mass of soft friable tissue which has a very thin shell of cortical bone. Microscopically it consists largely of large spindle cells and osteoblasts, with little tendency to ossify.

Prognosis and Treatment—In elderly patients amputation offers the best chance of cure; but in young adults less than 5% are curable by this means. Irradiation will control the lesion in these cases sometimes for several years but will not cure.

Tumours of Non-osseous Origin

Ewing's Tumour

Histogenesis—There is no proof of the origin of this tumour. The primary focus is specific for bone and is probably intracortical or possibly subperiosteal. There are some grounds for the belief that it is a primary lymphoma of bone.

Clinical Features—This is a growth of the first two decades of life which typically is first seen as a single tumour but which ultimately metastasizes widely to the other bones. Tibia and femur are most commonly involved primarily. An epiphysis is never involved except secondarily. There is a history of pain, tumour, and dysfunction occurring over about thirteen months. Constitutional symptoms are common.

X-ray—Typically there is a diffuse lesion near the mid-shaft which causes expansion of the shaft and stimulates the periosteum to lay down layers of new bone, giving rise to the so-called onion peel appearance. The cortex is thickened and there is mottling of the medulla. The tumour extends more readily in a plane parallel to the axis of the bone.

Pathology—The gross appearance is that of a soft disintegrating mass. Microscopically it consists of numerous small round cells with dark nuclei and resembles a lymphosarcoma.

Prognosis and Treatment—Irradiation should be used as a therapeutic test, but will not cure. Radical excision is necessary following the maximal effects of irradiation and this yields about 10% of 5 year cures.

Multiple Myeloma

Histogenesis—This is not known but there is a tendency for some writers to consider it to be a tumour of bone marrow somewhat akin to the leukemias.

Clinical Features—This is a disease of later life, 40 to 70. When first seen it is usually multiple, as the name implies, and involves typically the ribs, sternum, clavicles and spine. The tumours vary from a pin point to an orange in size. Pain is severe and characteristic. Tumour, deformity, and fracture may occur. Pulmonary changes and nephrosis occur in the later stages.

X-ray—There is a characteristic distribution of round punched out areas of bone destruction of variable size. Pathological fracture is common.

Pathology—There are multiple, soft, gray tumours of the marrow which have produced bone destruction. Microscopically the growth is composed of numerous cells of the plasma-cell type.

Prognosis and Treatment—The usual course is about two years. Growth is uninfluenced by any form of treatment but palliative treatment may be necessary.

Fibrosarcoma

Histogenesis—This tumour may arise from the non-osteogenic layer of the periosteum, or from the fascia of muscles or nerves.

Clinical Features—This is a disorder of adult life, above 30. It occurs mainly in the lower extremity and is characterized by the occurrence of pain, swelling, and dysfunction. Growth is rapid and metastasis to the lungs may occur.

X-ray—There is a single lesion of the lower limb with a large soft part shadow and which tends to invade and destroy the bone from without. There is little or no periosteal reaction.

Pathology—This is a firm, fibrous, encapsulated tumour consisting of fibrous tissue arranged in whorls or striated lobules. Under the microscope, fibrous tissue showing malignant features is seen.

Prognosis and Treatment—The prognosis is good following complete removal in the early stages. There is a strong tendency to run a slow insidious, inevitable course.

Miscellaneous Tumours of C.T. Origin

Neurogenic Tumours—These tumours arise from neurogenic elements in the tissues around the bones and invade the bones from without. The clinical and pathological picture is similar to fibrosarcoma but the prognosis is distinctly worse.

Angioma, myosarcoma, and lipoma may invade the bone from without and often cannot be diagnosed without biopsy.

Secondary Tumours of Bone

Symptoms—The features which usually call attention to the presence of metastases are pain, pathological fracture, or possible neurologic disturbances. Suspicion may be aroused from the presence of a primary tumour or other metastases.

X-ray—The lesions are multiple and most often involve the vertebrae and femora. There are two types of lesions.

The *osteolytic form*, typified by the secondaries of carcinoma of the breast, is evidenced by the presence of bone destruction with little or no evidence of repair and no periosteal reaction. The initial involvement is medullary and the cortex is destroyed from within.

The *sclerosing type* shows just what the name implies, a sclerosis or increased density of the bone, with evidence of increased periosteal activity.

Source—The primary may be in the breast, kidney, prostate, testicle, bladder, uterus, ovary, thyroid, stomach, lung, or more rarely in other situations.

Differential Diagnosis

Bone tumours have to be differentiated from a wide variety of conditions at various times. Such conditions include:

1. Blood dyscrasias—Hodgkin's disease, leukemia, chloroma, lymphosarcoma and Gaucher's disease.

2. Infections—Osteomyelitis, tuberculosis, syphilis, Brodie's abscess, Garre's

non-suppurative osteitis, or ossifying periostitis.

3. General conditions—Paget's disease, osteomalacia, osteitis fibrosa cystica, or myositis ossificans.

Conclusion

An attempt has been made to review the subject of bone tumours. The emphasis throughout has been laid on histogenesis, as the clinical features are a logical outcome of the mode of origin. The subject has been presented in rather a dogmatic fashion, but the reader must realize that the histogenesis of bone tumour is one of the most controversial questions in pathology, and hence any attempt to do otherwise would defeat the object of this paper.

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TUBERCULOSIS OF THE HIP-JOINT

S. KLING, B.A., '43

The purpose of this article is to serve as a guide for the *EARLY* diagnosis of tuberculous hip-joint disease. At the same time all the typical signs and symptoms are mentioned with their clinical significance. To complete the topic there is a brief discussion on the pathology of the condition and an outline of modern methods of treatment.

Occurrence

(a) *Frequency*—Of all bone and joint disease produced by the Tubercle bacillus, tuberculosis of the hip-joint is second in frequency only to Spinal disease, the approximate ratio being 7:10. It is the commonest of the joints proper to be affected.

(b) *Age*—The disease is most prevalent in children between the ages of one and six. It is not infrequently found in adults also. These adult cases are very apt to be mistaken for osteo-arthritis, at least in the early stages, and so may pass undiagnosed. Unfortunately adult cases offer much less resistance to the disease process than do the childhood cases with the result that adults offer a poorer prognosis.

(c) *Sex* seems to play no important role in the occurrence of this disease although it is slightly more predominant in males.

(d) *Race*—The racial susceptibility to hip-joint disease has a similar incidence to that of Pulmonary Tuberculosis. The resistance of the Indian, Negro and Mexican is notoriously low; the yellow race is slightly more resistant; and the white race has the most marked resistance of all.

Etiology

The infecting organism is the Tubercle bacillus, usually of either the human or bovine type and in rare cases, the avian type. Actual infection, as in all types of tuberculosis, is by direct or indirect contact with an open case of tuberculosis or by ingestion of contaminated food. Congenital infection is rare although there are two cases on record at the Fort Qu'Appelle Sanatorium which are believed to be of this type.

Predisposing factors are—

(1) *Poor general health.*

(2) *High resistance of soft tissue.*

(3) *Recent infectious fevers.*

(4) *Trauma*—Cases of tuberculosis of the hip often date the onset of symptoms from some trauma. Perkins et al consider trauma in such cases to be merely coincidental. Chandler and Mercer, in an excellent monograph, consider trauma to be an important localizing factor lowering the resistance of the injured tissue.

Pathogenesis and Gross Pathology

The joint lesion is always secondary to pulmonary, gastrointestinal, or glandular foci. As a rule there is a primary infection of lymphatic glands from which the bacillus spreads, via the blood stream or lymphatics, to the synovial membrane or the bone. In the very young the joint disease is usually secondary from a tuberculous focus in the articular end of the bone. In adults the primary synovial form is the commoner. In either case

symptoms suggestive of joint disease arise when the synovial membrane becomes infected.

In the common type, i.e. the small child with a primary bone focus, the bacilli are lodged near the epiphyseal cartilage of the head of the femur—a place where the circulation is active and the growing bone less resistant—with the formation of an area of infected granulations. Typical tubercles develop and grow circumferentially, destroying bone without stimulating reparative reaction and thus forming local abscesses. Extension to the joint proper results from—(i) destruction of the epiphysis and articular cartilages or (ii) by extension along the epiphyseal line to capsule and synovia. Regardless of the path taken the end result in an untreated case would be destruction of the cartilages by these granulations (pannus) which finally attack the bones of the joint. In the early stage there is a simple effusion but the fluid soon becomes infected with tubercle bacilli and the whole joint is invaded by the disease.

The synovial membrane becomes thickened, edematous, gray and ulcerated. The bones become denuded of their protective cartilage, are eroded and sequestra may form. As the disease progresses the head of the femur is partly absorbed, the remnants being dislocated from the acetabulum on to the ilium where a false joint is formed. This is constantly pushed upwards by the muscles acting on the head of the femur, giving rise to the so-called "wandering" or "migratory" joint.

At a later stage the pus which has formed bursts through the capsule and spreads in the lines of least resistance. It may perforate in the groin, in the neighborhood of the great trochanter, or by perforating the acetabulum, appear as a pelvic abscess.

Histopathology

In the early stages there is an infiltration of endothelial cells surrounded by lymphocytes. Later, groups of the endothelial cells coalesce to form giant cells of the foreign body type, surrounded by amorphous material. Occlusion of the blood vessels, aided by toxins, leads to disintegration and caseation. Bacilli can escape from the focus to form new tubercles which in turn coalesce and so enlarge the area of the disease.

Clinical Aspects

(a) Symptoms: (i) *Limp*—This is the earliest, commonest and most important symptom of the tuberculous hip. At first the limp is protective and may be so slight as to be indiscernible to all but the trained eye. It is best noticed in the early morning on arising from bed and again late in the evening when the patient is tired. Later the limp is due to other factors such as apparent shortening, deformity and eventually due to true shortening, the result of bone destruction.

(ii) *Pain*—This is a relatively late symptom and often is absent in patients seen within a short while of the onset of the disease. It is a gross and regrettable error to think that there must be pain before one can diagnose tuberculosis of the hip. If one waits for pain much valuable time is lost in commencing treatment with the result that the prognosis is much worse. Pain, when it does occur, may be either *local* or *referred*.

(a) *Local Pain* is a variable symptom. The severity of the pain is presumably proportionate to the intensity of the inflammation and also to the degree of tension within the abscess cavity formed

by the accumulation of caseous material. Thus if the site of the disease is in the bone and there is no outlet to the surface of the bone, pain might be expected to be severe. Likewise a rapid accumulation of fluid in the joint cavity would give rise to severe pain. On the other hand, where the caseous material is draining freely into soft tissues around the joint, pain is minimal.

(b) *Referred Pain* is the commoner of the two. This may be felt anywhere in the leg because all the main nerves of the limb supply branches to the hip. Perhaps the commonest site is the inner side of the knee.

While on the subject of pain it is necessary to mention *"night-cries."* This is where the victim while sleeping, gives rise to a piercing cry due to intense pain, and the patient may be awakened at the same time. A story of "night-cries" is often elicited from a patient with a tuberculous hip and therefore it is important to realize their meaning and significance. Their presence implies that the articular cartilage is eroded and thus a knowledge of their presence is an important guide as to whether this has occurred. It is of no value, however, in establishing an early diagnosis as one does not expect to get a history of night-cries in the early case any more than one expects, on examining the patient, to find a sinus. They are both associated with a late stage of the disease.

(b) Signs

General Examination—A general survey of the child should be undertaken although this is not of much value from a diagnostic point of view.

(i) *Temperature* will usually be found to be normal and the occasional rise is no more than can be accounted for by the excitement of examination. Here again it must be remembered that we are dealing with an early case and it is a mistake to expect to see a rise in temperature.

(ii) *Weight* — This should be recorded, not as a diagnostic aid, but for future reference. The actual weight may be above or below the average; it is the increase or decrease as observed over a long period of time which is important.

Local Examination—For this the patient should be naked. It not only facilitates examination but it renders less likely that the surgeon will fail to observe the presence of a second tuberculous focus. It can be a humiliating experience to discover suddenly, and by accident, a second well advanced focus of infection in a patient who has been under one's care for some time.

It is wise to have a hard couch and a routine system of examination. A sequence of examination which has proven satisfactory is the following. One looks for—

(i) *Deformity*—This should be looked for in all three planes. In the early stage there is flexion, abduction, and lateral rotation, because less weight is borne on the joint in this attitude and there is apparent lengthening of the leg. The flexion at the hip abolishes the normal gluteal fold. The pelvis tips down.

As the disease progresses, the joint is unable to tolerate any strain so that crutches become necessary and the attitude now is one of flexion, adduction, and medial rotation with apparent shortening. This is due to the stronger muscles overpowering the weaker; the hip-joint of a spastic paraplegic is similarly distorted.

As the bone becomes eroded and wasted the real shortening supervenes and increases as the dislocation of the head of the bones develops. There is in addi-

tion retardation of growth from disturbances of the epiphysis.

(ii) *Tenderness*—Local tenderness on palpation over the head of the femur is sometimes present and to my mind is indicative of an abscess under tension.

(iii) *Swelling*—Generally a fullness over Scarpa's triangle can be appreciated due to fluid in the joint. Some authors (Tubby and Kirmisson) state that the inguinal glands are always enlarged. Other authors, including the staff of the Fort Qu'Appelle Sanatorium, have not found this to be necessarily so.

(iv) *Abscess*—Considering the pathological process involved and the clinical and radiological evidence of spinal tuberculosis it is likely safe to assume that in tuberculosis of the hip a cold abscess is present. However in the case of the hip the abscess is buried deep among the muscles around the joint and takes some time to appear on the surface. If the abscess is under tension it will cause pain. Severe pain and local tenderness will indicate the presence of an abscess, even when it cannot be palpated. The site at which the abscess presents will depend on (i) site of its origin and (ii) gravity.

(v) *Muscle Wasting*—This is a sign of the utmost importance. The presence of muscle wasting, particularly of the buttock muscles, is of great value in diagnosing an early case. The cause of the wasting is not clear but it is evidently an active process rather than merely due to disuse as shown by comparison of the two limbs of any patient with hip disease after he has been treated on a frame for a year with both legs equally immobilized. A striking difference will be found in the degree of wasting of the hip muscles on the two sides.

(vi) *Limitation of Movement* — In tuberculous arthritis there is a limitation of movement in ALL directions, because the limitation is due to muscle spasm. In late disease scar tissue forms around the joint as a part of the healing process and this also produces a limitation of movement. Therefore it is sometimes difficult to tell whether the limitation in a given case is due to continued activity of the disease process or to adhesions around the joint.

(vii) *Shortening* — This has already been mentioned under deformity. Two measurements should be taken—(i) from the anterior superior iliac spine to the tip of the medial malleolus.—True shortening. (ii) From the umbilicus to the tip of the medial malleolus.—Apparent shortening. The difference between the true and apparent shortening indicates the position of the affected joint. When the apparent shortening is greater than the true shortening the limb is adducted. When the apparent shortening is less than the true the limb is abducted. It must be remembered that in taking the "true" measurement the limbs must be held in identical positions. The "apparent" measurement is taken with the legs parallel.

True shortening is not found at an early stage of tubercular arthritis and true shortening in the early stage of an arthritis would be against a diagnosis of tuberculosis. A word of warning is necessary, however. Sometimes two healthy limbs are not of the same length. Therefore when shortening does exist its site should be located. This can be done by testing whether the greater trochanter is raised above Nelaton's Line, and by measuring from the anterior superior iliac spine to the head of the tibia. If the great trochanter is raised above Nelaton's

Line, as compared to the other side, an amount equal to the true shortening of the limb, then the shortening is somewhere between the greater trochanter and the acetabulum which is compatible with tuberculous destruction of the head and neck of the femur.

Diagnosis

(i) The finding of some of the above signs and symptoms.

(ii) Tuberculin—As the majority of the cases of hip tuberculosis occur in children under six, a positive tuberculin reaction in the presence of the proper clinical findings should make one increasingly suspicious of tuberculosis. On the other hand, a negative reaction to a strong dose (one mgm. in children) is strong evidence against tuberculosis. Only 3% of patients with clinical evidence of tuberculous lesions give a negative reaction.

(iii) Radiographic examination—Radiographically, the tuberculous hip follows quite a characteristic course. *AT AN EARLY STAGE* nothing abnormal is noted. The earliest sign is a slight haziness due to synovial edema. In a few cases, owing to its excessive thickness, it is possible to delineate the synovial membrane above and below the head. With great effusion the ends of the bones are farther apart than on a normal side. *LATER* bone atrophy develops and the bone is somewhat rarified. The affected side gives a thinner joint shadow and still later an area of destruction is seen, most commonly in the so-called Babcock's Triangle, i.e. on the inferior aspect of the cervical side of the epiphysis.

FINALLY when the cartilage is eaten away and more and more bone eroded, the joint outline disappears. The erosion is most marked on the upper hemisphere of the head, and the upper part of the acetabulum.

The diagnostic value of radiography is not great in the early stages of this disease, but is useful at a later stage in indicating the site and severity of the lesion. Radiograms are used:

(1) As an aid in diagnosis.

(2) To locate the site of the disease.

(3) To determine the severity of the lesion.

(4) To demonstrate the cessation of activity of the disease.

Differential Diagnosis

There are several conditions which are apt to confuse the diagnosis. These will be grouped under their most prominent symptoms and signs, viz. limitation of movement, abscesses in the region of the hip, limp, and pain.

1. *Limitation of Movement*—In tuberculosis this is due to muscle spasm. Other conditions which will also produce this and which must be ruled out are:

(a) Subacute Mono-articular Non-Tuberculous Arthritis. This is not uncommon in children. If non-tuberculous it will disappear after a few weeks rest. The child should be examined every fortnight for a few weeks.

(b) Hysteria is recognized by the variability of symptoms, the marked hypertonus of the muscles, and the absence of wasting.

(c) Spastic Paralysis—Upper motor neurone lession—produces no wasting and usually there are signs of the spasticity in other part of the limb, e.g. Babinski, etc.

(d) In Reflex Irritation—as from inflamed glands, injured adductors, or

phimosis, limitation is not general as in tuberculous hip-joint disease.

2. *Abscesses in the Region of the Hip* —If the abscesses are acute there is little difficulty. If subacute the possibility of hip disease must be considered. If the abscess is tuberculous (as told by culturing aspirated pus) it must be differentiated from:

(a) Tuberculous Bursitis of the Subgluteal or Psoas Bursae—in which the limitation of movement is not general.

(b) Psoas Abscess spreading down from a spine infected with Pott's disease.

It is in these cases that a complete X-ray examination will be of value.

3. *Limp.*

(a) Congenital Dislocation of the Hip —is present from birth, the head of the femur is in an abnormal position, and one can produce "telescoping" of the limp. X-ray is decisive confirmation.

(b) Coxa Vara—Lateral rotation is increased and there is marked deformity. X-ray examination is of the greatest value.

(c) Pseudocoxalgia (Calvé Legg-Perthés Disease)—is a very hard distinction to make.

There is

—less muscle wasting than in tuberculosis;

—the movements of the hip are not limited in all directions as in tuberculosis;

—Radiological — (i) The bone is sclerosed rather than rarified.

(ii) The joint space is increased rather than decreased.

(iii) The head of the femur is flattened and occasionally fragmented, thus producing an apparent shortening of the neck. The whole thing resembles a "mushroom" in appearance.

4. *Pain.*

(a) Osteomyelitis—The main feature here is local tenderness and toxemia. A differential white blood count would show a polymorphonuclear leucocytosis whereas in tuberculosis there is a lymphocytosis.

(b) Slipped Epiphysis—The severity of the pain, eversion, shortening, and absence of wasting are suggestive. X-ray at once reveals the nature of the condition.

(c) Anterior Poliomyelitis — has a more sudden onset, often arising during an epidemic. The muscles are markedly hyperesthetic and tender. In a few days the joint can be moved freely in all directions.

Prognosis

With an early diagnosis and efficient treatment the mortality should be very low. Any undue intensity of a symptom is unfavorable. Age is also an important consideration, the danger both to life and to function being greater in adults.

As for impairment of function, Sir Robt. Jones states that "if the damage is sufficient to make the diagnosis of Tuberculous Hip-Joint Disease a certainty, then some impairment of function is certain." The degree of impairment varies with the site of the lesion.

In acetabular disease and that confined to the neck of the femur, if the treatment has been efficient, cure of the disease may be looked for with a half to a third of the normal range of movement.

When the head is affected the result is always doubtful and if there is much bone destruction, ankylosis in a good position is the best result that one can expect. In this variety there is a marked tendency to adduction even after treatment has been carried out for some time.

TREATMENT

1. *Prophylaxis* — As in all disease there is no cure like prevention. The details of prophylaxis are too numerous to be discussed here.

2. *General Treatment*—This is of the utmost importance in all types of tuberculosis. One must keep in mind that this is not an isolated skirmish between the hip and the germ but represents an invasion by the bacilli against the health of the individual. Therefore the individual's resistance must be kept at a maximum by

(a) good, nutritious diet.

(b) open air.

(c) sunshine.

3. *Local Treatment*—This will vary according to the stage and extent of the disease.

(i) *Stage of Acute Symptoms*—

(a) *Treat the Pain*—When the patient is suffering from acute pain in the joint, is unable to move without the greatest distress and is frequently awakened at night by pain arising from friction between the inflamed joint surfaces, no treatment gives speedier relief than traction. This is often applied through the medium of a Thomas' Knee Splint. To steady the pelvis and prevent compensatory adduction of the other leg a long Liston splint is applied to the opposite side of the body.

(b) *Correct the Deformity*—This is done by careful and gradual traction. It is sometimes recommended that cross traction be applied to pull the head away from close contact with the acetabulum. In practise, however, this treatment does not seem to have any marked effect.

(ii) *Stage of Cure*—When the acute symptoms, particularly pain and spasm, have subsided and the deformity has been treated, it is necessary to decide what our aim in treatment will be. The aim of treatment in this stage depends on the degree of destruction caused by the disease. We can divide all patients into two classes.

(a) *Those With Movement at the Joint*—These include cases in which the cartilaginous surface covering the head of the femur is intact, i.e., neck, acetabular and synovial disease. Such a case is treated by *traction* with the object of separating the articular surfaces and preventing bony ankylosis. If this attempt is unsuccessful any stiffness that remains is due to fibrous adhesions and the range of movement will depend on their length. For such a case a Pyrford Hip Frame is used. It is an ingenious combination of the ordinary Bradford Frame used for spinal cases, with two Thomas' knee splints fixed to the frame at the level of the hip joint. These knee splints are moveable to permit adjustment of abduction.

(b) *Those With No Movement at the Joint*—These comprise cases in which the cartilaginous surfaces have been damaged and the bones allowed to come into apposition. If the head of the bone is much eroded ankylosis is bound to ensue. It is therefore wise, in such cases, to aim at complete and firm ankylosis. In the majority of cases this is necessary.

Ankylosis can best be obtained by immobilising the hip with Plaster of Paris. There is an art in applying a plaster splint so that the maximum of immobilisation is achieved with a minimum of pressure. To master this art requires much skill and experience.

The above are the two ways by which the hip is treated in the second stage. When the disease is no longer active the convalescent stage begins. Unfortunately

it is very difficult to say when activity ceases. If the local signs of disease have disappeared and if the patient has a good appetite, is gaining weight, and has no fever, the disease is probably quiescent. X-rays are not of great value, but they may show that the appearance of decalcification is being replaced by that of more normal density. The practical test is to cease local treatment temporarily and to watch the response of the joint to freedom from treatment. The tractions are released and the leg allowed to move about—the patient still being kept recumbent. If the disease is active the muscles will reassert their protective action and prevent the joint from moving, i.e., they will go into a state of spasm. The freed joint is examined daily. If, as a result of freedom, either the joint gains an increasing range, or, in the absence of an increase in movement, the muscles do not go into spasm when a forced movement is attempted we assume that activity has ceased and that the convalescent stage of treatment may be entered (Thomas-Gauvain Test).

(iii) *Stage of Convalescence* — The disease is now arrested and rehabilitation of the patient is the next consideration. Usually for the next two or three months the patient is allowed freedom in bed unhampered by any splints or apparatus. He is then allowed up with a walking caliper. A patten is fixed to the boot on the sound side and crutches are used. The patient is usually advised to wear his caliper for at least a year, during which time he should be re-examined at regular intervals. The patten is removed after about six months and the crutches discarded three months later. After all apparatus has been discarded he is asked to report every three months for three years.

4. *Operative Treatment*—A surgeon must not forget his limitations. The surgeon belongs to the Salvage Corps and not to the Fire Brigade. He stands by and sees that as little damage as possible is done during the process of cure; he does nothing to assist in putting out the fire. His duty is to see that when the disease becomes quiescent the joint is in the best possible condition for subsequent function.

However, it has been abundantly proved that there is also a place for operative treatment which may be justified for two reasons.

(i) In many cases conservative treatment does not cure the disease but produces a state of chronic invalidism.

(ii) In many favourable cases it takes a very long time for recovery to be effected.

The four main indications for operation are:

(i) In an adult patient.

(ii) Failure of conservative treatment to arrest the disease after one to two years.

(iii) Relapse, especially the recurrence of pain and deformity after conservative treatment.

(iv) Certain destructive lesions, e.g., the formation of sequestra in the head or neck of the femur, or in the acetabulum.

The operation may be directed to the treatment of the primary disease (primary operations) or to the improvement of the function of the limb which has been the seat of the disease (secondary operations).

(A) *Primary Operations*—

(i) Excision of the Head of the Femur—This is done in an attempt to remove all the tissue infected with the tubercle bacillus. A study of six cases in which this operation was performed reveals uniformly bad results.

(ii) Arthrodesis—This operation may be performed is various ways but the aim in all cases is to produce a firm, bony ankylosis by (a) removing the articular cartilage of femur and acetabulum, (b) bone grafts, (c) a combination of (a) and (b). A detailed discussion of the surgical technique would be out of place in an article of this type, but one of the best known techniques is that developed by Dr. R. I. Harris of Toronto. Arthrodesis is inadvisable before the age of 10 years, and even in older patients, unless there is definite disease in the bone. The requirements for successful arthrodesis are:

(a) An operation which shall be mainly extra-articular in order to avoid the difficulties attendant upon free opening of the joint.

(b) A substantial graft of high osteogenic power in intimate and extensive contact with both femur and ilium.

(c) A small gap to be bridged by the graft.

(d) Firm fixation and maintenance of the graft in its bed until fusion has occurred.

(B) *Secondary Operations*—To correct an old fixed adduction-flexion deformity some form of osteotomy is usually employed.

(i) In cases of sound bony ankylosis with adduction-flexion deformity a subtrochanteric or a trans-trochanteric osteotomy may be performed and reduction produced.

(ii) In some cases the disease is cured only after the head of the femur has become dislocated. In these cases one may attempt to replace the head in the original acetabulum and abduct the leg as described above. In many cases the head cannot be brought down into the original acetabulum. In such cases a modified Lorenz's osteotomy is practised. Here the operation is carried out about the trans-trochanteric region in an oblique upward and inward direction. The leg is then abducted and the inner sharper end of the lower fragment is slid into the region of the acetabulum while the outer aspect of the upper end of its shaft fuses to the lower end of the proximal fragment.

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SCHIZOPHRENIA

G. LILIAN HUTTON, R.N., '46

Schizophrenia is the newer term applied to the group of mental disorders formerly known as dementia praecox. The older term has been replaced because:

(1) It overstressed the pessimistic outlook of the disease.

(2) It was a misnomer in that the disease, though ocurring chiefly in youth or early adult life, may and frequently does develop in the third or fourth decades. Schizophrenia is essentially "a cleavage of the intellectual processes and the emotional responses". It is marked by progressive emotional deterioration and is, in effect, an escape mechanism of a maladjusted, unsound personality.

Four types of schizophrenia are described:

(1) Simple.
(2) Hebephrenic.
(3) Catatonic.
(4) Paranoid schizophrenia.

However, the differentiation is not sharply marked and the terminal stages may all show much similarity.

In the *simple* type the patient exhibits a growing indifference to his personal appearance and hygiene. From the world of reality and responsibility he escapes into his private realm of fantasy. Though he overindulges in day dreaming, he does not suffer from hallucinations or delusions. This is the most benign form of the disease and frequently ends in spontaneous recovery.

Hebephrenia is marked by hallucinations of a vivid nature. These may affect any of the special senses. Peculiarities of behaviour and conversation result from the combined effect of emotional deterioration and hallucinations. Onset of hebephrenia after forty is unfavourable for eventual recovery.

The distinctive feature of *catatonia* is negativism. While in the catatonic stupor, the patient is unclean in his habits, often refuses food, may be mute, frequently exhibits suspension of all voluntary activity. This stuporous stage is usually preceded by or alternates with periods of excitement. The prognosis for catatonia is relatively good.

Paranoid schizophrenia typically comes on later in life (40 - 45). It progresses slowly and is marked by delusions (usually of persecution) and ideas of reference. Intellectual deterioration is frequently absent and, in such cases, systematized delusions of an amazingly plausible nature are voiced. Occasionally such a patient appears in court and his mental integrity may be so well preserved as to mask thoroughly the emotional defect. A miscarriage of justice may result. When, as sometimes happens, this type of schizophrenia develops atypically, in youth or early adult life, it is malignant in character.

Prevalence

The following figures were obtained from the 1941 report of the Ontario Provincial Hospitals. They may be taken as indicative of the general occurrence of schizophrenic illnesses. Of the 14,314 patients under hospital treatment, 6,493 (45.29%) were suffering from schizophrenia. Of the 2,541 first admissions for the year, 567 (22.31%) were schizophrenics. The rate per 100,000 population is 171.9 for incidence, and 14.9 for first admissions.

Prognosis

The relatively gloomy prognosis and the tendency toward chronicity are suggested by the discrepancy between admission and incidence rates. Despite new and improved methods of treatment, 50% - 80% do not recover; 60% deteriorate; 50% of cured cases relapse. Even this dark picture, however, is an improvement over the abysmal outlook of not so many years ago.

Factors Modifying the Prognosis

Much research has been done on the possibility of attacking schizophrenia via the "back door" method of a better understanding of the factors affecting its development and modifying its prognosis. As a result of this work the following information is available:

(1) *Age:* Except in the atypical onset of hebephrenia after forty, and of paranoid schizophrenia before forty, age bears no direct relationship to prognosis.

(2) *Duration of Illness:* This appears to be a positive factor, the prognosis varying inversely as the duration of the illness.

(3) *Previous Attacks:* When there is a history of previous attacks, the prognosis improves directly with a lengthening interval between the attacks.

(4) *Heredity:* "Susceptibility depends on specific type of predisposition which is single factorial, autosomal and probably recessive although modifiable in its expression by effect of mesodermal defense mechanisms. The individual capacity for developing protective and compensatory reactions to the morbidifying tendency of any schizophrenic genotype seems genetically determined as a graded non-specific character that is multi-factorial and produces a wide range of variations in the vulnerability and the constitutional resistance to the original predisposing factor.

As soon as we shall be able to duplicate or sufficiently reinforce the heredo-constitutional mechanisms responsible for a satisfactory resistance to the disintegrating effects of a schizophrenic process, the therapeutic problem of schizophrenia will be nearer its final solution." (Kallemann and Barrera.)

(5) *Body Type:* The asthenic constitution is most susceptible to schizophrenia. Strong resistance is exhibited by athletic types. A pyknic constitution appears to preclude development of the disease. Kretschmer found that, in atypical cases where schizophrenia developed in conjunction with a pyknic type, the prognosis was unfavourable.

(6) *Personality Type:* It is the introvert or schizoid type which predisposes to schizophrenia. Where the disease occurs in an introvert personality combined with an asthenic constitution, the outlook is ominous.

(7) *Onset:* Sudden onset is more favourable for recovery than is a slow insidious development of the disease.

(8) *Precipitating Factors:* Schizophrenia precipitated by environmental conditions or by faulty mental activity has a brighter prognosis than that resulting from constitutional and inherited factors alone.

(9) *Confusion:* The prospects for favourable termination of the disease depend upon the degree to which consciousness becomes clouded. Paranoid schizophrenia with its well preserved mental processes is most malignant, while catatonia with its great confusion demonstrates the most favourable outcome.

(10) *Treatment:* With modern treat-

ment, many cases formerly considered hopeless can be cured or improved sufficiently to carry on with a fair degree of economic and social usefulness.

Treatment

All forms of treatment are aimed at prevention of deterioration, correction of poor habits of reaction and modification of inherited tendencies. Persistent strong stimulation is an essential part of any therapeutic measure. A "total pull" co-operation of doctor, hospital staff and family to effect a "total push" against the malignant disintegrating process of the disease is the ideal environment in which to treat schizophrenia.

ROUTINE THERAPY

Symptomatic measures as indicated by the individual case should always be the immediate treatment:

(1) *Medical or surgical treatment* of underlying chronic infections or diseases.

(2) *Hydrotherapy* as indicated to combat toxemia, catatonia or excitement.

(3) *Massage* should be considered when practicable.

SHOCK THERAPY

Unless age, general ill health or organic heart disease contraindicate, some form of shock therapy is usually tried. The favourable results obtained in many cases justify this drastic therapy despite the occasional fatality. There are three types of shock treatment in use, namely:

(1) *Insulin shock therapy* consists of the production of a series of shocks by gradually increasing doses of insulin over a period of six days, followed by day of rest. The customary reaction is profound coma; convulsions may occur. After a definite period of coma, the patient is revived with glucose solution. This treatment is continued for weeks or months, as indicated by the patient's response.

(2) *Metrazol shock therapy* consists of bi-weekly rapid intravenous injection of metrazol. Violent convulsions follow immediately upon injection of the drug. The danger of fracture is ever present. Consciousness is regained in about ten minutes. This treatment is continued over a period of five to ten weeks, and, after a rest interval, may be followed by second series of injections.

In the last three or four years it has been demonstrated that 43.1% of metrazol patients suffer multiple fractures of the thoracic vertebrae. But, considering the valuable results of the therapy, and since the fractures clear up leaving no clinical symptoms, the discovery has not been accepted as a contraindication for treatment.

(3) *Electric shock therapy* is still experimental. It has the definite advantage over metrazol of eliminating the fracture hazard. Whether it will prove to be of as positive value as the latter, time will reveal.

SURGICAL THERAPY

In limited and definitely chronic schizophrenic cases where all therapeutic measures including shock treatment have been tried with no effect, prefrontal lobotomy has been undertaken with favourable results. The operation is resorted to only after every other possibility has been exhausted. This hesitancy is induced by the fact that operation results in a permanent destruction of the prefontal pathways and

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the patient is left with certain post-operative defects—they tend to develop habits of gluttony, poor self control and a distorted self appreciation. However, encouraging results have been obtained in cases of long duration. Recovery (with negligible defect) has occurred in several cases in which before operation had been extreme deterioration.

Summary

1. Schizophrenia is a disintegrating process which upsets normal behaviour patterns and emotional responses. It may or may not be associated with mental deterioration. Hallucinations or delusions, or both, may be present, but not necessarily.

2. The disease is essentially one of maladjustment with heredity and constitution providing a variable background of predisposition.

3. The prognosis, though poor, it not as hopeless as was formerly believed.

4. The chronicity and malignancy of schizophrenia justifies the risks involved in drastic therapy where the latter has, even in moderate degree, brightened the prognosis.

5. Psychiatric research and mental hygiene are two angles from which the problem is being attached, by studying and attempting to modify the predisposing and precipitating factors responsible for initiating the schizophrenic process.

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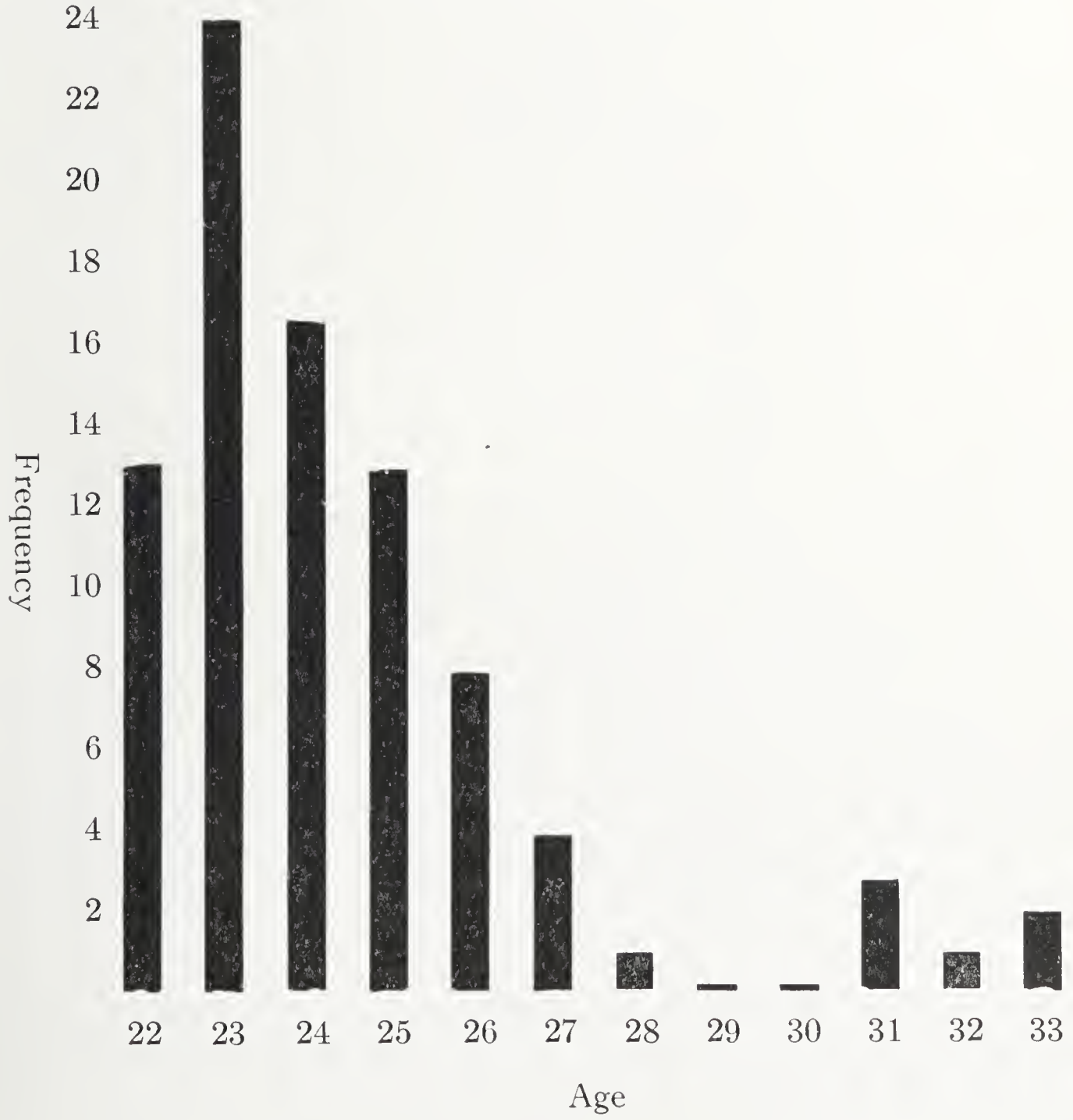
Feature Section

During the past year the Feature Section has been reserved for articles which, though not of as technical a nature as those in the rest of the Journal, have been of general interest. These articles are usually by an expert in the field under discussion. Such articles are planned for this year, and will appear in later issues.

* * * * *

A survey was carried out in October among members of the graduating class. Certain facts become apparent, as one examines the results. Eighty-two members of the class of 110 answered the questionaire. This group may be considered representative of the whole class.

Mean Age : *24.6*
Modal Age : *23*
Median Age : *23-24*



The average age of the class is 24.6 years; the modal age is 23. Thus, with the possible exception of theological students, medical students are the oldest group in the University. Even under the best conditions, they will have passed close to half their lifetime preparing for their vocation.

Further, we see that 18 of this group, or 22% have married, some recently, many not so recently. This is an unprecedented figure, yet the fifth year bids fair to exceed it. As a general rule, doctors and other professionals marry at a later stage. The high incidence of married medical students is only a reflection of a trend in the general population.

It is difficult to state how many doctors each of the armed services will require—of those who answered, the following preferences were expressed:

NAVAL MEDICAL SERVICE	15%
R. C. A. M. C.	41%
R. C. A. F.	44%

More than 80% of doctors are general practitioners. It is difficult, indeed, for a student to be certain of what he wishes to do, and in addition, no one can predict the result of the force of circumstances. However, the expressed desires of the students are illuminating:

General Practice	27
Surgery	21
Obstetrics and Gynecology	7
Internal Medicine	6
Pediatrics	4
Public Health	3
Otolaryngology	2
Psychiatry	2
Biochemistry	2
Anesthesia	1
Ophthalmology	1

An extremely interesting finding, though one difficult of interpretation, was the way in which the class answered the question, "If you had to do it all over again now, would you enter Medicine again?" The surprising result was that 10 students, or over 12% answered "No." Very few stated what they would have prefered to taking the course, though certain members expressed preferences for engineering, forestry, journalism, and chemistry. Of the rest, 67 answered "Yes," and five could give no definite answer.

Thirty-nine students or 47% claimed to have some technical or professional training, other than medical. Surveyors, photographers, pharmacists, machinists, and chemists were the occupations most frequently listed. Some were farmers and sailors.

Conclusions

No general conclusions can be drawn from so small a sample. However, we invite the comments and suggestions of our readers regarding the results, and regarding the survey in general.

Abstracts

The Application of Phlebography to the Therapy of Thrombosis and Embolism

By Claude E. Welch, M.D., Henry Faxon, M.D., and Claude E. McDokey, M.D., Boston, Mass.

Increasing clinical interest in the problems of thrombosis and embolism has led to the frequent use of phlebograms both for diagnoses and to furnish necessary pre-operative information. In the last year phlebograms of over 100 lower extremities have been made by various members of the peripheral vascular clinic. Seventy-three of these cases have been checked anatomically because of exploration and legation of the femoral vein.

Methods of Injection: Twenty-five cc. of diodrast are injected into the short saphenous or dorsal veins of the foot and at the conclusion of injection the X-ray exposure is taken. To ensure injection of the deep veins return of the blood through the long saphenous vein is prevented by use of a tourniquet. Thrombosis of this vein is detected easily clinically.

Experience has shown that the phlebogram is not absolutely normal in so far as diagnosis of thrombosis is concerned and a normal phlebogram with positive clinical signs of thrombosis should be considered to have thrombosis.

Indications:

1. In any case of known or suspected thrombosis of the veins of the lower leg.

2. After a pulmonary infarct unless it unquestionably arose elsewhere than the leg.

Many patients require bilateral phlebograms and they may be taken one after the other without risk.

The application of this procedure is to furnish information not demonstrated clinically, and certain groups of cases are discussed which demonstrate the value of the phlebogram. The inaccuracy of clinical examination in any case of thrombosis is well known and many previous studies of thrombo-embolic problems have been entirely misleading since the venous systems involved have not been noted accurately.

The diagnosis of thrombosis is important even though no specific therapy is contemplated. This is seen in the occasional patient who post-operatively develops a fever and elevated pulse. After ruling out other possibilities the diagnosis of thrombos-phlebitis may be made by a phlebogram.

Pulmonary infarcts have occurred with no evidence of the source. Phlebograms of the legs may show the source of the emboli and ligation of the femoral vein will result in cure.

An acute superficial thrombophlebitis is present. Phlebogram is indicated and frequently an associated widespread thrombosis of the deep venous system will be found.

The phlebogram is useful for the early detection of deep thrombosis when suspected and early therapy is contemplated.

Many different types of pictures are obtained in these cases.

Therapy: The control groups of this clinic showed one infarct out of every three cases of deep phlebitis and the death rate of patients over 50 years was nearly 8% because of massive infarcts alone.

Eighty-two femoral veins for deep venous thrombosis were ligated, the average age being fifty-six. According to the control group with conservative treatment twenty-five pts. would have had further pul. infarcts and six would have been fatal. After legation only two infarcts occurred, neither being fatal. The site of ligation is important and determined by the phlebogram.

P. H. Spohn.

Salicylate Prophylaxis in Rheumatic Fever

The Journal of Pediatrics, Aug., '42

By Alvin F. Coburn, M.D., and
Lucille V. Moore, M.D.

Clinical observations on the effectiveness of acetyl salicylic acid in preventing serum sickness and in vitro studies on the interference of antigen-antibody precipitation by salicylates stimulated the idea of the application of salicylate prophylaxis to rheumatic children living in overcrowded tenements of New York City and exposed to prevalent respiratory infections. This report is the result of two years' study.

The clinical manifestations of rheumatic disease bear a close similarity to those of serum sickness. Clinical observations have shown there are three distinct phases in the development of acute rheumatism. Phase 1 lasts a few days during which time the patient has an upper respiratory streptococcal injection. Phase 2 is a symptom free incubation period commonly about ten to fourteen days in duration. Phase 3 is the rheumatic attack. At the end of phase 2 there is diminution in serum complement suggesting an antigen-antibody reaction. Phase 3 is accompanied by the appearance of circulating precipitins. It was shown that poly arthritis occuring in serum sickness was prevented by the administration of salicylates and patients so treated failed to develop a marked rise in precipitins whereas untreated cases did not escape arthritis and had a rise in precipitins.

In the group studied there were one hundred and eighty-six young rheumatic subjects. At the onset of pharyngitis salicylate therapy was instituted and throat cultures obtained. If cultures contained hemolytic streptococcus Group A, prophylaxis was continued for four weeks (daily dose of 4-6 grams). If another pathogen was isolated salicylates were discontinued (none of this group developed rheumatic fever). The control group was made up of cases who failed to notify their condition of pharyngitis. Blood was taken once or twice a week for two months for determination of the sedimentation rate and antistreptolysin titre.

The results were that the groups of forty-seven rheumatic subjects treated with sodium salicylate following hemolytic streptococcal pharyngitis escaped clinical manifestations of rheumatic fever with one exception. Fifteen of this group shared a brief asymptomatic rise in sedimentation rate within a few days after prophylaxis was discontinued. Thirty-one maintained normal sedimentation rates.

Fifty-seven of the 139 in the control who contracted pharyngitis developed rheumatic fever. Six types of identified streptococcus organisms were responsible for most of the infection in both the

treated and untreated cases; that is the groups were infected by the same serologic types of Group A, streptococcus hemolyticus. Sodium salicylate did not modify the antistreptolysin response. The discontinuation of salicylates four weeks after infection showed a rise in sedimentation rate on fifteen patients but with exception of one the development of rheumatic activity was suppressed below the clinical margin.

These observations prove that rheumatic fever can be prevented by the administration of salicylates during the respiratory infection and silent phase. Also that the association between salicylate phophylaxis and the failure of rheumatic subjects to develop attacks following streptococcal pharyngitis is significant.

P. H. Spohn.

Eclampsia

By Kenneth W. Sewall

New England Journal of Medicine, Aug. 20, 1942

Theories of etiology, physiologic pathology, pathology and treatment are reviewed.

The cause remains unknown in spite of many theories. One suggestion is that placental infarcts and degenerating placentral tissue liberates autolysates which if massive or if renal function is poor, produce severe late toxaemia or eclampsia. It is believed that the infarcts are due to cholesterol-induced arterial disease. It seems that whatever its cause, eclampsia affects first or even arises from the placenta. The chief pathologic damage is to the vascular system, and possibly it may have a hormonal origin.

Concerning pathology, belief is expressed in the conception that arteriolar derangement, affecting all the terminal arterioles is the primary factor in the disease. On this basis is explained the degenerative renal changes to which has been given the term glomerulonephrosis. The liver lesions of haemorrhage, necrosis and fatty degeneration are likewise placed on a vascular basis. Other changes described are adrenal cortical haemorrhages, splenic haemorrhages, generalized arteriolar spasm, and ischaemic cellular disease of the brain.

The most frequent signs and symptoms before convulsions were albuminuria, hypertension, oedema, headache, visual disturbances, vomiting and pain. Blood uric acid is elevated and a decrease in CO_2 combining power is noted, great emphasis being placed on these latter findings as indicative of impending convulsions.

The usual nursing routine in conservative treatment is followed. Sedatation is important. 50% glucose i. v. is recommended to decrease oedema, increase urinary output and combat acidosis. 20 c.c. of 10% mag. sulph. i. v. is given at each convulsion and between convulsions if the blood pressure starts rising. Venesection is advised if the above fails to lower blood pressure, 200-400 c.c. of blood being removed.

When convulsions are controlled, and the patient appears improved, or if the patient does not improve and it is desirable to avoid further delay, the membranes are ruptured. If pelvic delivery is contraindicated, Caesarean section is justified.

G. J. Quigley.

Complications of One Stage Abdominoperineal Resection of the Rectum

Thomas E. Jones, M.D.

Journal of the Amer. Med. Assoc.

This is a review of over 500 cases of one stage abdominoperineal resections for

cancer of the rectum and the various complications arising from such are enumerated and discussed.

(1) *Parotitis:* Appeared in only 5% of cases, the process being frequently mild and only occasionally proceeding to suppuration requiring incision and drainage through the capsule of the gland.

(2) *Pulmonary Complications:* These were found to be in direct relation to the incidence of infections in wounds post-operatively. In an early group, in which there was some degree of wound infection in 28% cases several deaths were due to pneumonia. Following the institution of interrupted alloy steel wire sutures in the closure of abdominal wounds, infection dropped to 1% and with it the incidence of pneumonia. Atelectasis was uncommon because of its lowered tendency to occur in lower abdominal operations. At its first appearance, intravenous sodium sulphathiazol was given in an endeavour to prevent pneumonitis in the collapsed areas. Tracheal catheterization is also of value. Pulmonary embolism showed a smaller incidence in spite of the magnitude of the operation. The majority appeared 10 days following operation. There was not one case of femoral phlebitis and its sequelae in all these cases.

(3) *Peritonitis:* This was the foremost complication years ago and the chief cause of death. It was invariably associated with an infected wound and seemingly secondarily infected from it. The symptoms usually appeared about the 4th or 5th day and were not fulminating from the time of operation and as the peritoneum can throw off infection more readily than the wound, such must have come from deep wound infection. When wounds were closed with catgut, infection occurred in 28% cases and mortality varied from 9 to 12%, whereas in cases using interrupted alloy steel sutures with simple clip closure to the skin and the elimination of stay sutures, wound infection occurred in 0.85% of cases with a mortality of 4.5% and only 1 death in the last 82 consecutive cases. Neither intraperitoneal vaccine or sulphonamides were used. Paralytic ileus is uncommon and when it appears it is generally due to mild peritonitis or plastic exudate due to various causes. Death from haemorrhage is rare. Post-operative haemorrhage during the first 24 hours occurred only occasionally, it being generally due to oozing from vessels over the prostate or upper part of the vagina and responds to further packing for pressure. Shock was not observed, the use of spinal anathesia and routine transfusion during operation eliminated.

(4) *Infection of the Urinary Tract:* These constituted the most distressing complications. Because of wide pelvic dissection and the destruction of sympathetic fibres the physiology of micturition is destroyed which so predisposes to infection. Cystitis in some degree occurred in 95% cases and limited to the bladder clinically, chills and costovertebral tenderness occurring only rarely. In those who can void spontaneously the symptoms are mild. The fever of this complication appears about the 5th or 6th day and lasts usually 4 days following the use of chemotherapy depending of course on the persistence of residual urine.

(5) *Epididymitis and Damage to the Ureter at Operation* were rare; prolapse of a loop of bowel through the pelvic floor was not seen; late obstruction occurred once; neoplasm in the remaining colon occurred three times; impotence due to destruction of sympathetic nerve fibres

cannot be prevented in this radical operation.

Renal Pathological Changes in Hypertension and Glomerulonephritis: Clinical Interpretation

J. P. Simonds, M.D. J.A.M.A.
Vol. 120, No. 2. Sept. 12, 1942

An excellent presentation well worth the attention of those engaged in clinical medicine and most especially to those who are attending closely to pathological chemistry. It is a straightforward discussion with clinical observations interpreted in light of the pathology found.

G. G. C.

Genu Recurvatum Following Poliomyelitis

A controlled method of Operative Correction.

C. E. Kerwin, M.D.
J.A.M.A., Sept. 26, 1942

There are two separate and distinct types of genu recurvatum which follow an attack of poliomyelitis, the presence or absence of structural changes in the tibial condyles serving to differentiate them. Such changes, or their absence, can be explained by the variation in the underlying causative factors. In the first group is the lack of sufficient power in the quadriceps to lock the knee in extension against resistance. The changes that account for hyperextension are found in the condyles and upper third of the tibial shaft. The former are elongated posteriorly and in the latter there is a posterior bowing and in many cases a partial subluxation is present. This deformity develops slowly over a long period of time according to the law that "the change in static relations of bone leads to changes in structure and physiologic function."

In the second group, weakness in the calf and hamstring muscles is the underlying cause of the deformity. Here the hyperextension develops rather rapidly. Elongation of the above muscles followed by stretching of the posterior capsular ligaments so allows the joint to hyperextend.

Surgical procedures designed to correct the deformity must fulfil two important requirements. First, the mechanical alignment of the hyperextended extremity must be restored and second, following such, the underlying causative factors of the deformity must be so altered so that the deformity will not recur from the original mechanism. The prognosis following surgical intervention in the two types is entirely different. In the first group, it is excellent, and in the second group extremely poor such that a long brace is eventually necessary to keep these extremities in good alignment. The operation which is discussed in this paper is concerned with the correction of the hyperextension and not mith the secondary procedure that is necessary to alter the underlying causative factors. It is an operative procedure of comparative simplicity, consisting of an osteotomy of the fibia and fibula and the proper controlling of these fragments.

G. G. C.

Myocardial Infarction

Clinical Features and Prognosis

H. W. Rathe, M.D.
J.A.M.A., Vol. 120, No. 2

The material presented consisted of 274 cases of myocardial infarction seen during or shortly after what was considered to be the first attack and in which the diagnosis was confirmed by electrocardiograms.

These cases, for study, were divided into 3 groups. (1) Early fatal group who died within one month of the onset of their first clinical symptoms. (2) Late fatal group who lived longer than one month. (3) Survival group, various factors which were believed to influence this condition were so investigated with the following results.

(*a*) Age factors. That the patients who died were older than those who survived was shown by the fact that 74% of the fatal group were over 55 years of age at the time of infarction, whereas only 63% of those who survived had reached that age. Thus a poor prognosis was indicated when the age of the patient was over 55 years.

(*b*) Occupation. It was found that coronary artery disease was just as frequent in individuals who lived outdoors a great deal or who lived in rural areas as those in business or in the professions.

(*c*) History. A family history of hypertension, cerebral vascular disease or cardiovascular disease was found in 75% of cases. Hypertension was known to have been present in 63% prior to their first infarction.

(*d*) Exciting factors. Unusual exertion or excitement were important but found in relatively few cases.

(*e*) Symptoms. Severe pain was the most frequent major symptom of infarction lasting from 1 to 48 hours and in some patients continuing as a residual pain up to two weeks. Paroxysmal dyspnoea was another major symptom, usually accompanied by pain, but the most serious complaint. Unusual fatigue and apprehensiveness were pronounced in their occurrence while acute pulmonary oedema and shock were often present. Antecedent symptoms were dyspnoea, with or without exertion, Angina of effort, undue fatigue and epigastric distress or pain, not related to effort, in order of frequency of occurrence.

(*f*) Clinical observations. (1) When the heart rate was 100 or over during the first week and continued at this rate a poor prognosis was given due to the fact that 76% of the early fatal group showed this sign.

(2) Shock and muffling of the heart sounds occurred consistently in all groups but were valuable only in so far as diagnosis was concerned.

(3) Gallop rhythm was found in 51% of the early fatal groups.

(4) The subsequent development of cardiac failure was found most frequently in the fatal groups and was of importance in the rehabilitation of the patient.

(5) The development of congestive heart failure occurred in 42% of the early fatal cases and was of serious prognostic significance.

(6) In cases in which the systolic blood pressure dropped suddenly and failed to rise again to a level of 100 mm. Hg. in 5 days, a poor prognosis was given. Further a pulse pressure of 20 mm. Hg. or less was significant.

(*g*) Functional capacity. In group 2, 64% lived 1 year or longer but only 11% resumed normal activity. In group 3, 91% lived 1 year or longer and only 23% resumed previous normal activity. However, in this group activity in most cases was restricted therapeutically.

G. G. C.

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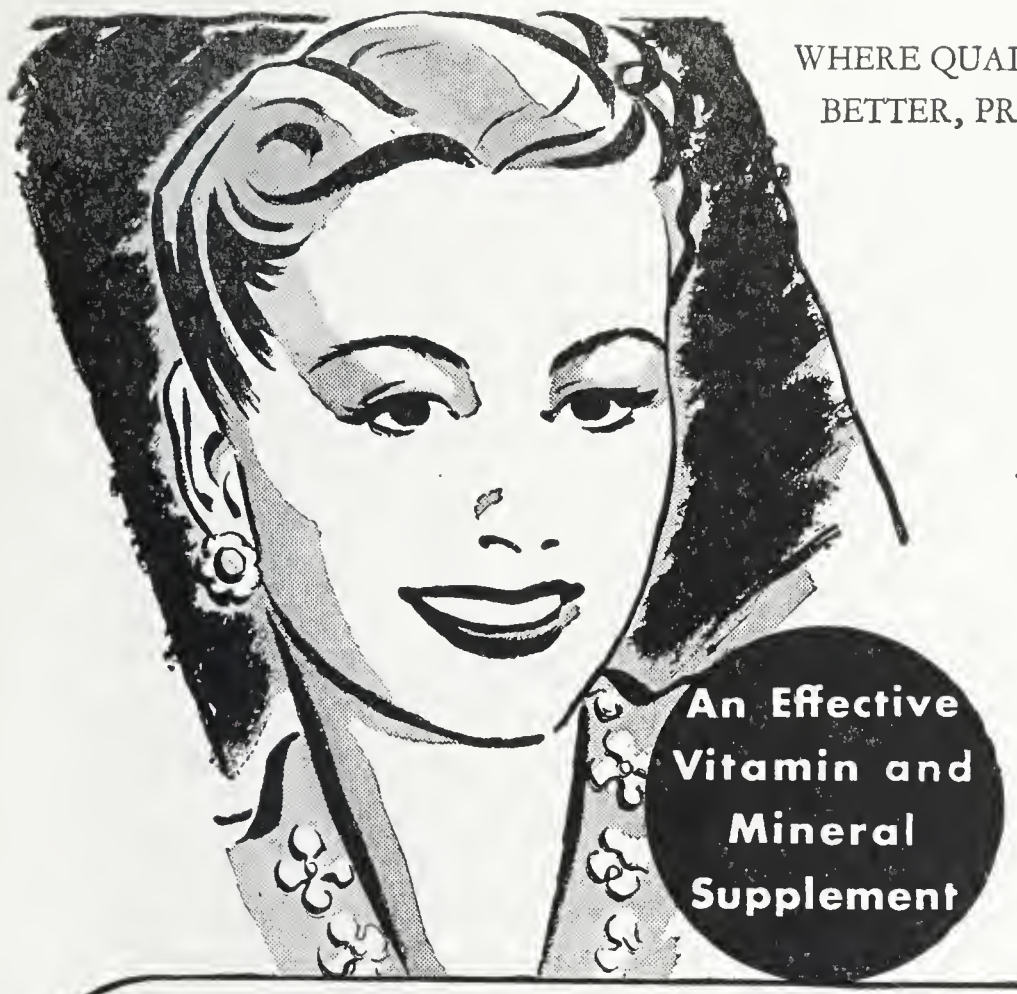

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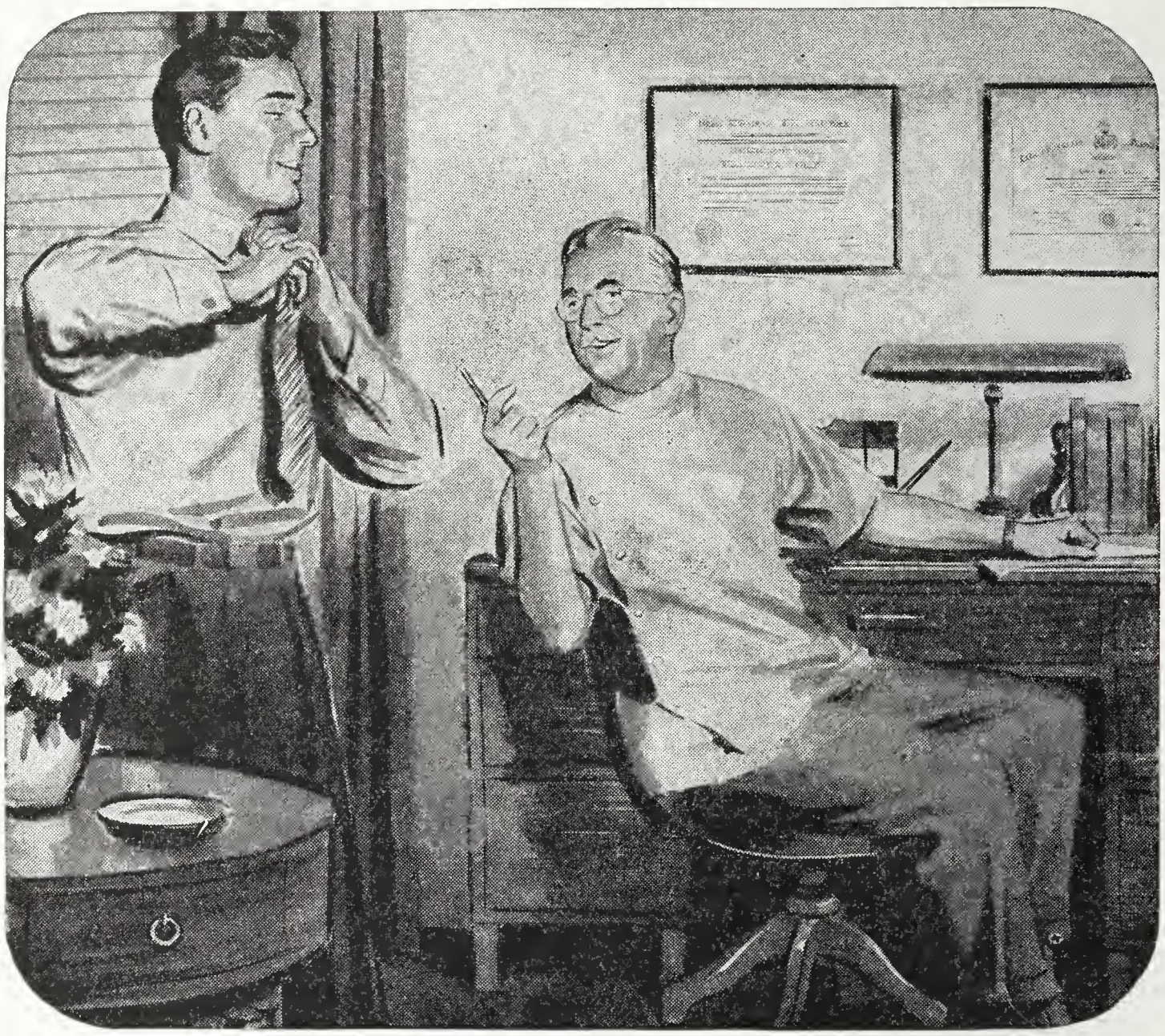

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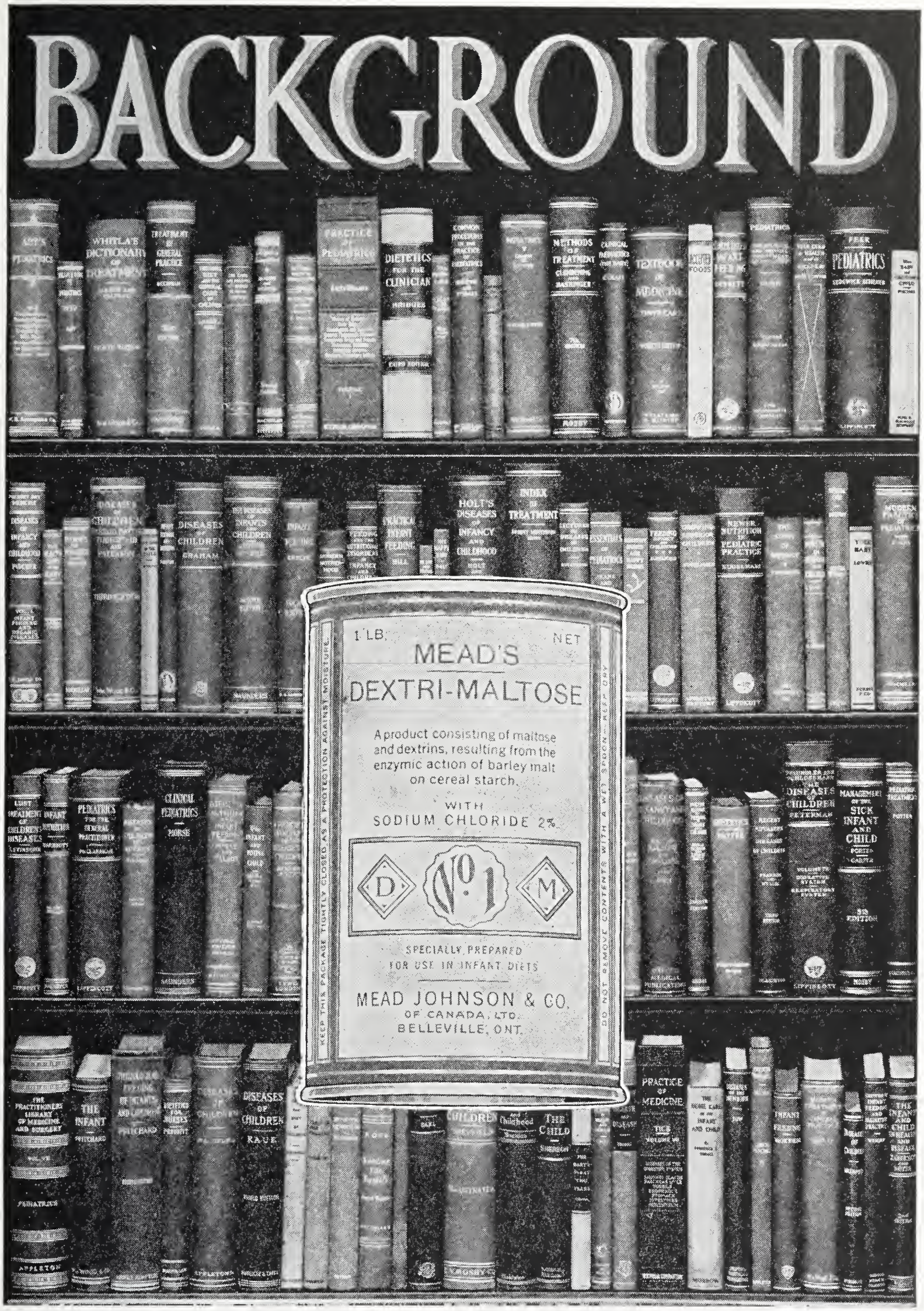

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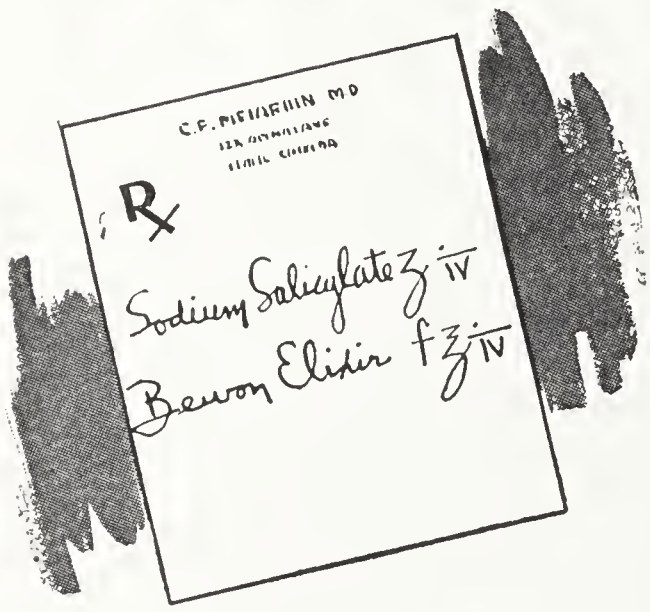

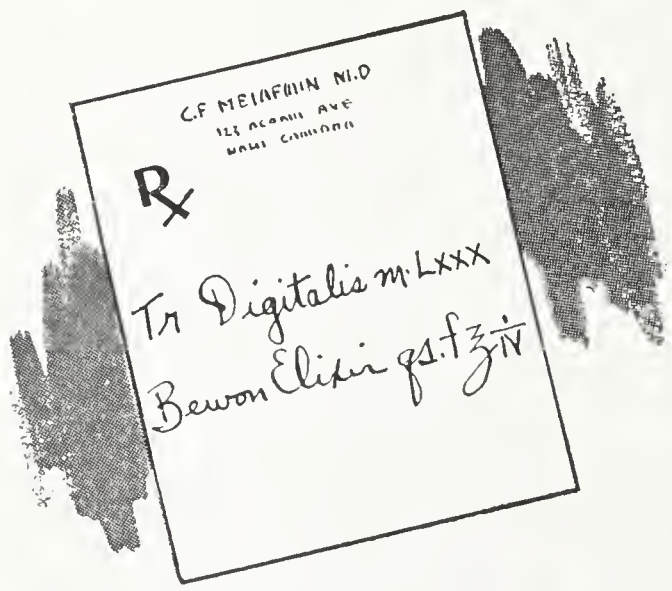

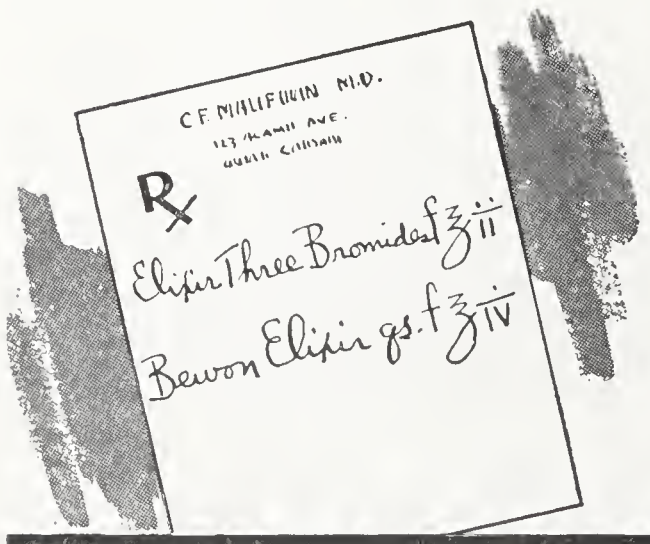

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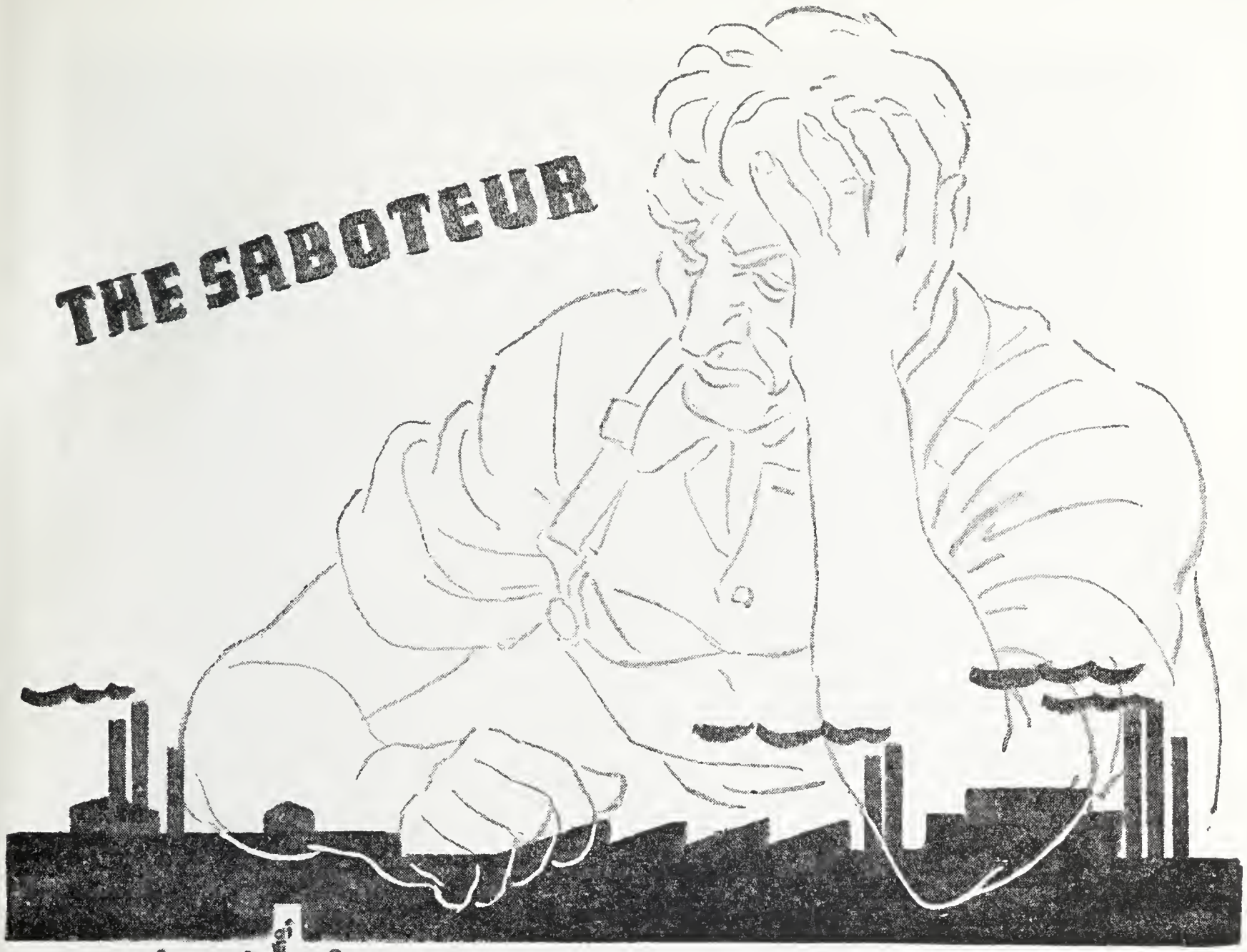

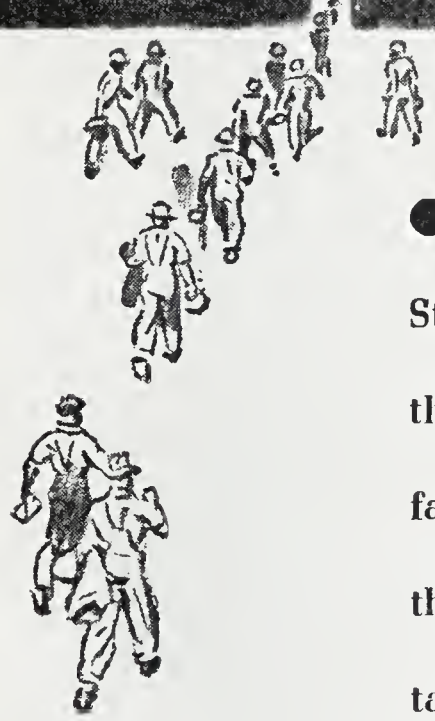

● Absenteeism due to illness is retarding our war effort. Study of the underlying causes reveals that, in many cases, the diets of employees are deficient in certain vital food factors. An extensive effort is now being made to improve the national diet but such a programme of public education takes time. Until the importance of proper diet is more generally understood, some supplementary means of providing essential vitamins and minerals should be employed. A daily dose of "Alphamin" can do much to improve the health of Canadian workers and so help to maintain war production at its highest level.

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- PRECIPITATES AND COAGULATES BACTERIAL SUSPENSIONS
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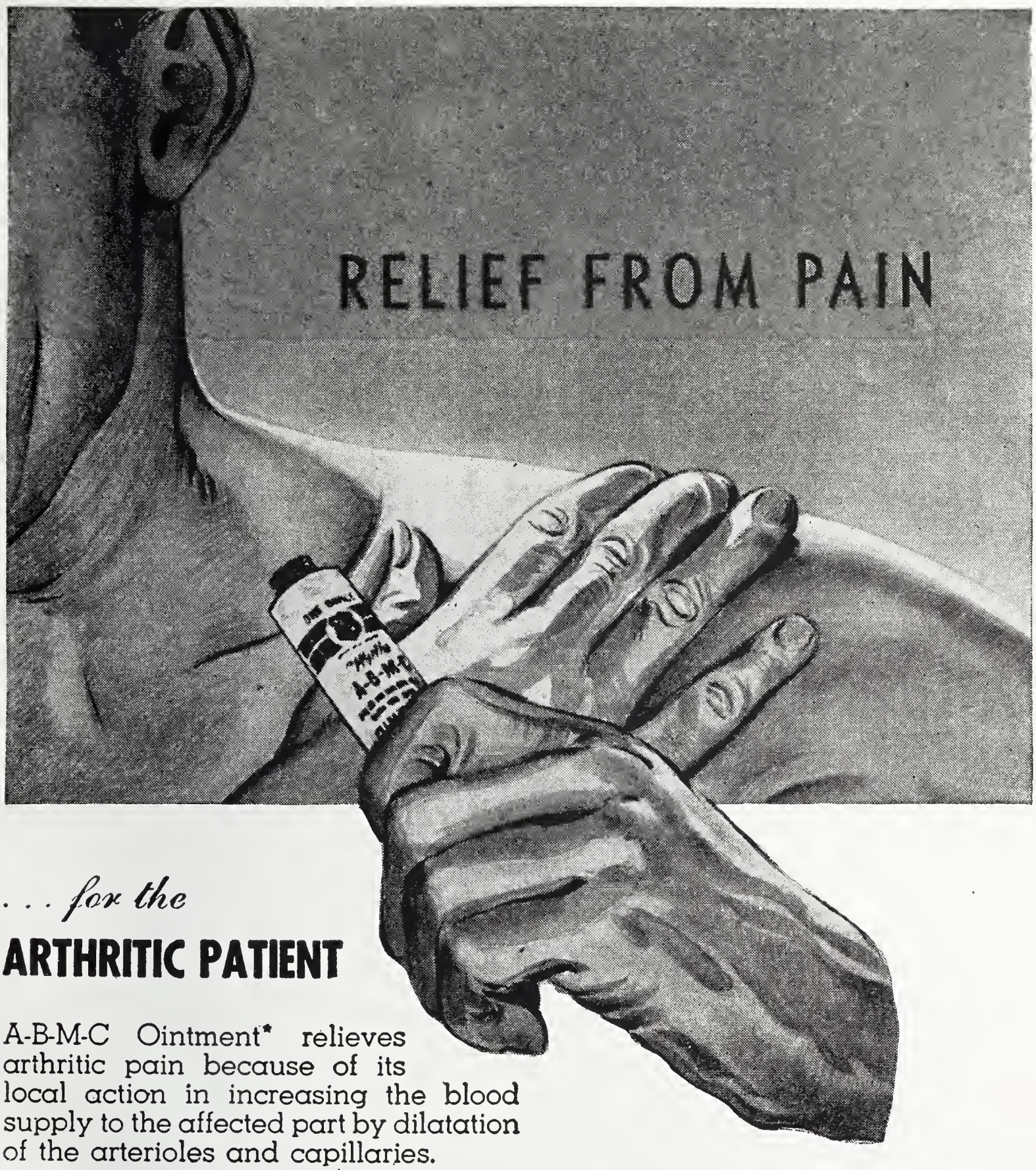

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A-B-M-C Ointment is spread, without rubbing, on the affected part and heat is applied for 20 minutes.

†Archives of Physical Therapy, *21*. 12 (Jan.) 1940.

**A-B-M-C Ointment is a trademark of John Wyeth & Brother (Canada) Limited, for its brand of ointment containing Acetyl-B-Methylcholine Chloride 0.25%, menthol, thymol, eucalyptol and methyl salicylate in an emollient base.*

Supplied in 1-ounce tubes

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(ACETYL BETA METHYLCHOLINE CHLORIDE)

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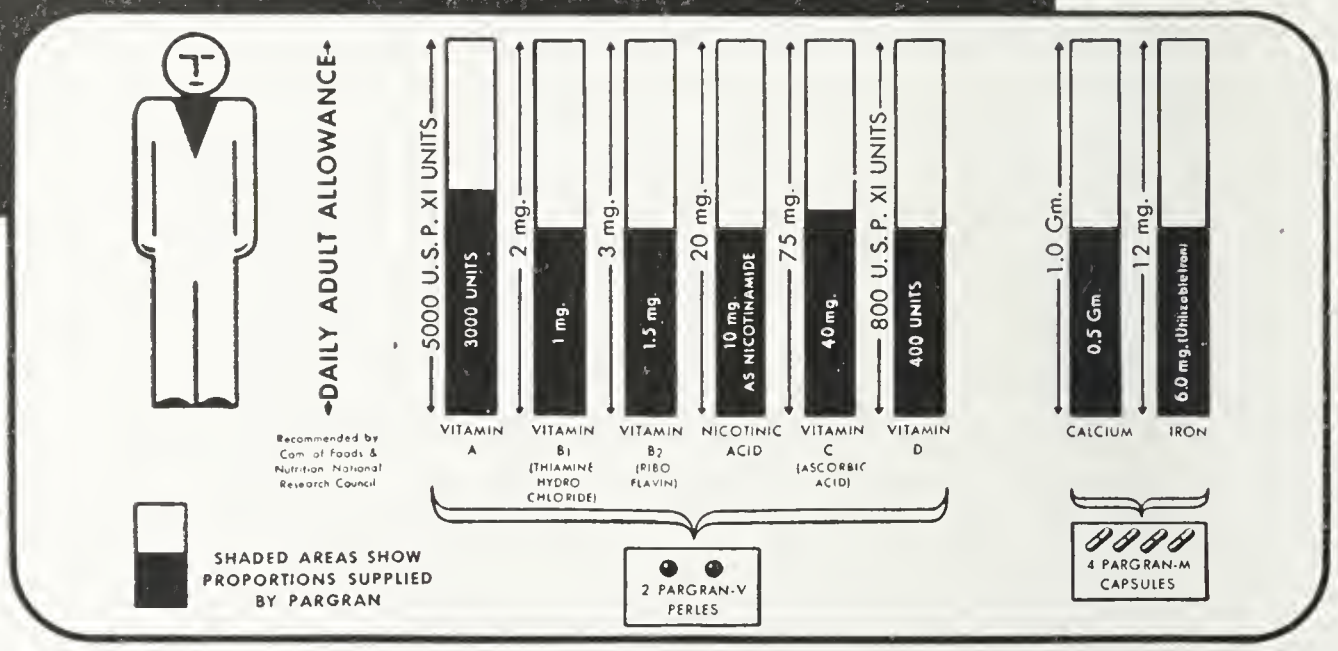

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